A SOLUTION TO THE PROBLEMS OF PAIN

by

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ABSTRACT

In my thesis, I challenge existing philosophical and scientific accounts of pain to explain certain constitutional, functional and empirical problems. Though difficult, some of these problems will be familiar. Unlike perceptual experiences, pains are strikingly affective and when we are in pain we are primarily concerned with the experience itself rather than mind-independent objects. The obvious explanation, that pain is not a perceptual experience, would be appealing if it were not for the fact that pains vary in quality, intensity and location. These seem to be features of paradigmatic perceptual experiences. The magnitude of another problem, the weakness of the correlation between pain and the stimulus, has been under-played. It is widely observed that those with chronic conditions experience pain in the absence of the stimulus and in circumstances like sport and war severe injuries occur in the absence of pain. I argue that the variable relationship between pain and the stimulus is normal; it is not confined to abnormal cases or circumstances. As no existing account proves explanatorily adequate, I develop a novel position called 'near-motivationalism' from a revisionary approach to the conceptual models of pain science. This has the power to solve the problems I set.

For my Dad –
who would have been very proud.

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INTRODUCTION

In this thesis I present a new explanatory account of pain that I call 'near-motivationalism'. The main features of this account are that a subject experiences pain when an affective mental state is attached to a token phenomenal quality drawn from the quality spaces that subserve either innocuous thermal or mechanical sensations. This makes the phenomenal quality feel as if it were intrinsically unpleasant and consequently it motivates its subject to behave in ways that prevent or minimise injury.

It may be questioned why another account of pain is needed. Pain science has developed a detailed functional, anatomical and physiological understanding of pain and, where pain science is lacking, a plethora of philosophical accounts complement the science by providing various perspectives on the subjective nature and function of pain. Barring a major scientific finding, one would be forgiven for thinking that there was little original to say about the subject. The problem I have identified is that theorists have largely ignored a significant source of empirical data.

Understandably, problem pain conditions – pains that do not resolve over time – are the major motivation for much pain science. These conditions impose an enormous economic burden on individuals and society, but more importantly they cause a great deal of suffering. The development of an understanding of these problem conditions is

crucial for effective treatment. This is not to say that 'normal' pains are ignored. On the contrary, a model of normal pain is the yardstick against which problem pains are measured. This model is informed by tightly controlled experimental paradigms that are relatively simple to interpret and reproduce. Consequently, models of normal pain are models of experimental pain.

This would not be a problem if the causes of pain in experimental settings were like the causes of pain in everyday, non-experimental settings. On the face of it there is some similarity. Stimuli used in the experimental setting, often high intensity thermal energy delivered by thermode or laser, are like the high intensity events involved in stubbing a toe, pricking a finger with a needle or grabbing the metal handle of a hot pan. The trouble is that these experimental pains are not representative of all normal or 'adaptive' pains. Often the causal influences on pain are much more complex. In particular, patterns of pain change dramatically in the presence of tissue damage. For example, someone with an injury will often begin moving the injured body part quite tentatively and report significant pain, but as they continue to move the pain diminishes even though the movements increase in magnitude. In these cases the tissue damage is constant while the intensities of pain and activity vary in an inverse relation to one another. It is also common for the opposite to occur, that is for pain to increase as the intensity of activity decreases. Crucially, these patterns of pain facilitate recovery so they cannot be passed off as abnormal, 'maladaptive' experiences or problem pains. As the sensory models that dominate pain science and philosophy cannot explain normal pain experiences like these, there is a powerful motivation for a new explanatory account of pain. However, the capacity to explain

these normal pains does not guarantee explanatory adequacy. A position with this capacity might be internally inconsistent, contradicted by other data or fail to explain other aspects of pain. The aim of this thesis is to determine which theory, whether near-motivationalism or its opponents, provides the best explanation for certain crucial aspects of pain including normal pains.

THESIS STRUCTURE

My thesis is divided into three parts. In Chapter 1, I set out six explanatory problems. In Chapters 2 and 3, I consider whether existing philosophical and scientific accounts can deal with these problems and conclude that they cannot. Consequently, in Chapters 4, 5 and 6, I develop a new account and explain how it solves the problems of pain.

Chapter 1 is devoted to the six problems that I present as a challenge to philosophical and scientific accounts of pain. These problems can be roughly subdivided into constitutional, functional and empirical categories. Philosophers have long been interested in the nature and function of pain. When we are in pain we tend to focus on the experience itself. We may stop doing something, take a pill, seek medical attention or respond in some other way, but in almost all cases our objective is to rid ourselves of the pain. The reason for this is simple: pain is unpleasant. This marks pain out from visual, auditory, olfactory and other experiences. These latter experiences may focus our attention on the bustle of the market, the shouts of the

stallholders and the smell of the food but they lack an intimate connection with a specific affect. Recently, some philosophers have become interested in empirical evidence that pain is constituted by affectively neutral and affective mental states. This explains the asymmetry between the subjective qualities of pain and other experiences, but it raises further constitutional and functional issues. What is the nature of the two components of pain, and why are they almost invariably experienced as if they were an homogenous whole? These interesting philosophical questions are central to understanding pain and they underpin the constitutional and functional problems I will present.

Evidence of pain in the absence of the stimulus, the occurrence of the stimulus in the absence of pain, and the variable relationship between the intensities of pain and the stimulus are a major, perhaps *the* major, focus of pain research because they are taken to be abnormal circumstances. The normal pains I trumpet above are also examples of the variability of the relationship between the stimulus and pain. Whether normal or abnormal all these circumstances require explanation.

Chapter 2 is focused on existing philosophical solutions to these constitutional, functional and empirical problems. The philosophical accounts I consider are representative of the broad naturalising trend within the philosophy of mind, but more importantly, the majority take into account evidence that pain is a composite experience. Given the space available to me, I have not been able to consider the merits of each account in detail. Instead, I have taken the liberty of dividing these accounts along generic lines into three competing positions. 'Perceptualism' is the

thesis that the affectively neutral and affective components of pain have a perceptual function; 'motivationalism'is the thesis that both components have a motivational function; and mixed 'perceptualism/motivationalism' is the thesis that the affectively neutral component has a perceptual function and the affective component has a motivational function. Although each of these accounts has explanatory advantages, I argue that none has the power to explain all of the constitutional, functional and empirical problems set out in Chapter 1.

The scientific account I consider in Chapter 3 is a hybrid theory developed from the multidimensional, pain matrix and nociceptive concepts of pain science. Roughly speaking, these concepts concern the function, the functional anatomy and the peripheral neurology of pain respectively. Taken individually, none is equipped to deal with all six explanatory problems. I conclude that these concepts are ambiguous, inconsistent, and explanatorily inadequate. Consequently, I take a version of the mixed perceptual/motivational theory reinforced by aspects of these conceptual models of pain science to be the best of the existing accounts of pain. However, this account does not address all of the constitutional, functional and empirical issues I set out in Chapter 1.

It is a striking fact that none of these existing accounts can explain many normal pain experiences. My diagnosis for this problem is the mistaken assumption that noxious intensities of energy are detected by sensory receptors. This assumption underpins the almost universally accepted nociceptive concepts of pain science. In Chapter 4, I argue that sensory receptors cannot detect noxious stimuli and use this as the basis for

a further argument that harmful or potentially harmful intensities of energy are discriminated by a central nervous system mechanism. These are the key arguments in my thesis. They enable me, in Chapter 5, to solve several constitutional and functional problems and to develop a position, which I refer to as 'near-motivationalism', that is consistent with these arguments.

Near-motivationalism takes pain to be an output of a system that has the function of preventing injury and promoting recovery. This system has components that evaluate whether intensities of energy pose a threat and whether pain is an appropriate response. In Chapter 6, I argue that the complexity of evaluating the former provides an explanation for the normal pains that are overlooked by existing philosophical and scientific accounts. Taken as a whole, my thesis currently offers the best explanation for several important problems of pain.

1 THE PROBLEMS OF PAIN

Anyone setting out to explain pain faces several difficult problems. Perhaps the most obvious of these is the asymmetry between pain and perceptual experiences like vision and audition. First pains, unlike visual and auditory experiences, are strikingly affective. Specifically, the unpleasantness of pain moves us. Second, when we feel pain we are primarily concerned with the experience itself, yet when we talk of seeing or hearing we are concerned with mind-independent objects like tomatoes and trains. The obvious explanation, that pain is not a perceptual experience, would be appealing if it were not for the fact that pains vary in quality, intensity and location. These seem like features of paradigmatic perceptual experiences. Relatively recently some philosophers have engaged with evidence that, despite appearances, pains are composite experiences constituted by an affectively neutral and an affective mental state. This evidence explains why pains feel unpleasant, but it raises difficult questions about the nature and function of pain and its constituents. Another problem, the overwhelming evidence of a weak correlation between pain and the stimulus, has been less widely recognised by philosophers, but it is an obstacle for theorists whatever their stripe. The challenge is to provide an account of pain that adequately overcomes these obstacles.

This chapter is divided into two main sections. In the first, I clarify what I mean by 'pain'. The second section, which is focused on the problems of pain, is divided into five sub-sections that can be roughly divided into constitutional problems (sub-sections *1.2.1*, *1.2.2* and *1.2.3*), functional problems (*1.2.4*) and empirical problems (*1.2.5*). In sub-section *1.2.2*, I present an outline of a syndrome called 'pain asymbolia' that has attracted the attention of several philosophers in recent years. This syndrome provides insight into the constitution of pain but it also raises a number of questions and creates significant taxonomical problems. So this sub-section is critical to understanding my use of terms in this thesis.

For the purpose of both simplicity and brevity, I have rather crudely divided philosophical accounts of pain into perceptual, motivational and mixed perceptual/motivational accounts. These divisions are intended to be broad and uncontroversial. They represent what I consider to be the most viable philosophical accounts of pain. A commitment to perceptualism or motivationalism about pain is nothing more than a commitment to the position that pain is exhausted by either its perceptual or its motivational function. The mixed position is that pain has dual perceptual and motivational functions.[1] As the evidence provided by those who are unable to experience pain is that pain is necessary for the motivation of behaviours that avoid or minimise tissue damage, perceptualism is the position that pain fulfils a necessary perceptual role in a system that yields these behaviours. Motivationalism is the position that pain fulfils a necessary motivational role in these behaviours and the

[1] I have borrowed the term 'motivationalism' from Colin Klein (forthcoming) though not his commitments, which are much more demanding than my simple characterisation. I discuss Klein's motivationalism in chapter 2, section *2.2*.

mixed theory is a combination of the two. These competing positions are set out in detail and analysed in chapter 2.

1.1 PAIN: THE TARGET OF MY THESIS

My aim, to provide an explanatory account of pain, may not strike everyone as needing disambiguation and justification, but some conflate pain and suffering and others argue that the pain that is picked out by our ordinary concept is not worthy of serious attention.

It is part of our normal linguistic practice to use the *words* 'pain' and 'suffering' synonymously even though PAIN and SUFFERING are distinct concepts.[2] To see this consider that talk of the pain of a pinprick is consistent with the denial of suffering; the pinprick caused pain but the pain did not cause suffering.[3] The distinction between these concepts is often expressed by the terms 'physical pain' and 'psychological pain' (or 'mental pain'); a subject can be in physical pain without having psychological pain and *vice versa*. In my view, these terms suggest a misleading dualism. *Both* pain and suffering are psychological[4] in the sense that they are

[2] This ambiguity is particularly evident in C. S. Lewis' book "The Problem of Pain" (1940) which is about suffering. It is also evident in David Biro's "The Language of Pain" (2010).
[3] My point is that *talk* of 'the pain of divorce' or 'the pain of losing a loved one', does not show that the experience of pain and the experience of suffering fall under the same folk *concept*.
[4] This is the view of the International Association for the Study of Pain (IASP) who provide a subjective characterisation of pain; pain is "always a psychological state" (IASP, 2014). The IASP's view carries significant weight because their taxonomy (the source of this quotation) has been developed by and with the help of some of the most world's most eminent pain scientists, including John Bonica, Harold Merskey, John Loeser, and Rolf-Detlef Treede.

experiential mental states;[5] only you have access to your pains and suffering. The two concepts are linked by the capacity for pain to cause suffering. Presumably this is why we express suffering (whatever the cause) by using words like 'pain', 'painful' or synonyms for pain like 'hurt' or 'ache'. So my thesis is targeted at the subjective phenomena picked out by everyday locutions like "My knee hurts" and "Her back pain's preventing her from working at the moment", rather than the phenomena picked out by "He's been hurting since she left him" and "His death was so painful for me". In other words, this thesis is focused on accounting for the phenomena that are tracked by our ordinary concept of pain.

Some philosophers argue that the ordinary concept of pain is either internally inconsistent or inconsistent with science and is not worthy of serious consideration.[6] I have little to say about the strengths and weaknesses of these arguments, except to point out there is a clear sense in which all these philosophers have it wrong. The phenomena that we identify as pain under our ordinary concept are the *explananda* of pain science.

[5] Note, I am *not* saying that pain is psychological *rather than* physical. I am saying these are inappropriate *terms* as both refer to subjective experiences.

[6] In particular Don Gustafson (2000, 2005) argues that the ordinary concept should be ignored, Valerie Hardcastle (1999) that it should be eliminated, and Christopher Hill (2009) that it should be radically revised. To be fair to these philosophers, each refers to particular contents; Gustafson to the content 'pain is a single quale'; Hardcastle to the content 'pain is a simple sensation'; and Hill to the contradictory contents 'pain is not representational' and 'pain is representational'. None of these philosophers considers the essential role that the ability to identify experiences as pain plays in pain science. It is worth adding that with the exception of the concept that pain is not representational, I am not convinced that any of these contents have folk status. See also, Alex Byrne (2012), Richard Chapman et al (2000), Ron Resnik (2000a, 2000b) and Kenneth Sufka (2000).

Typically, in human pain studies researchers correlate a stimulus[7] with a subjective report of pain. If a report of pain were the only thing linking these experiences, pain science would have no substantial anchor and a chaotic neurological picture would have emerged. This has not happened. The particularity of the anatomy and physiology that scientists have associated with pain provides an objective link between pains and it supports the intuition that pains are characterised by distinct phenomenology. However, it would be a mistake to assume the truth of this intuition. The design of many of human pain experiments is such that there is little reason to doubt that the pains evoked in these experimental settings are identified by their characteristic feel, but this does not imply all pains are identified by their phenomenology or that there is a characteristic phenomenology for pain. One of the key reasons for developing a taxonomy (in sub-section *1.2.2*) is that I want to leave open the possibility that some genuine pains lack the characteristic felt quality of experimental pain and so might not be recognised as pain. Additionally, it is possible that some experiences with this characteristic feel are not pain even though they might be identified as pain by their subjects.

The relationship between pain and the ability to recognise pain figures significantly in this chapter and in chapter 5, section *5.3*. The reason for this is that there is a trend towards taking our recognitional abilities at face value; to accept that those experiences we recognise as pain are pain and *vice versa*. This trend is evident in the taxonomy of the International Association for the Study of Pain (the IASP). They urge, "If they regard their experience as pain and they report it in the same ways as

[7] Most commonly in these paradigms, the stimulus is thermal energy delivered by a thermode or laser to the skin at a particular bodily location.

pain caused by tissue damage, it should be accepted as pain" (2014).[8] It is also there in the accounts of philosophers who have considered pain asymbolia. There is a good intuitive reason to follow this trend; when I introspect, my experiences of pain are distinct from other experiences. The apparently unique phenomenology of pain explains our recognitional abilities. As I mentioned at the end of the preceding paragraph I do not want to assume that pain has characteristic phenomenology, but I also need something to ground my use of the term 'pain'. In lieu of any viable alternative our recognition of pain is a good place to start.[9] My take on recognition is that conceptually mature individuals hold a concept that enables them to recognise pain. I call this the 'recognitional concept of pain':

> *REC* A subject holds *REC* if and only if she is *able* to categorise an occurrent experience as pain by reference to her memories of experiences she has learned to categorise together as pain.

Three points are particularly important; first, *REC* is an ability rather than a concept with a definitional structure; second, *REC* does not require the ability to *label* experiences as 'pain'; and finally, possession of this ability implies both intra- and inter-subjective consistency. Additionally, *REC* does not carry a commitment to the view that the experiences we could identify as pain under *REC* are united by

[8] Whether "acceptance as pain" should be interpreted as a tacit acknowledgement that an experience that a (conceptually mature) subject recognises as pain *is* a pain is another matter.

[9] The IASP define pain in terms that seem to conform to a recognitional understanding; pain is "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (2014). Although it is not entirely clear precisely what this means it represents a subjective understanding of pain, because they also write "Pain is always subjective" and "[pain] is always a psychological state" (IASP, 2014). Aydede makes much the same point (2005a, pp4-5). Alternative objective positions developed by Hardcastle (1999) and Hill (2009) are complex and controversial (see fn.6 above) and would not be suitable.

characteristic phenomenology. It does not imply that all of the experiences we could identify as pain under *REC* are pains or that all pains could be identified as pain under *REC*. In short, *REC* does not amount to an identity claim, namely pains are the experiences we could identify as pain under *REC*. Despite this latter denial, for the sake of the clarity of my account I will begin by taking it that pains are the experiences we could identify as pain under *REC*.[10]

1.2 THE PROBLEMS OF PAIN

The problems of pain can be roughly divided into constitutional (P1 and P2), functional (P3) and empirical categories (P4-P6).

P1 *Prima facie* pains are constituted by characteristically unpleasant qualities that vary in intensity and location.

P2 Pains have dissociable affectively neutral and affective components.

P3 Those unable to experience pain suffer a greater number of and more significant injuries than normal subjects.

P4 Pains are often experienced in the absence of the stimulus.

P5 The stimulus is often present in the absence of pain.

P6 There is a poor correlation between the intensity of pain and the intensity of the stimulus.[11]

[10] This is formally stated in *T* in section *1.2.3*, this chapter.

[11] I am not suggesting this is the only problematic pain related data. For example, Aydede reflects that when we are in pain our attention is drawn to the experience rather than something mind-independent (2009). Although my account of pain provides an explanation of this matter, I do not consider it to be a

P1-P6 can be construed as individual problems. As such each, with the probable exception of some of the problem cases that fall under P6, has at least one existing explanation. For example, P1 is explained by the evidence that pain is constituted by two mental states (it is explained by the evidence for P2), and scientists explain some cases of P4 in terms of the plasticity of neurological systems and processes. The question is whether any single account can explain all these problems. In chapter 2 I argue that no existing philosophical account has the explanatory power to address all these problems. Given their resources it may seem that pain scientists are best placed to explain these problems but in chapter 3, I argue that the concepts of pain science are somewhat inconsistent and cannot deliver such an account. This is of major importance to my thesis. In particular, the concept of a nociceptive system provides support for the view that pain is (at least partly) a specific sensory modality. It provides the support for, in philosophical terms, the position that pain is a representation or awareness of harmful intensities of energy. I analyse the concept of a nociceptive system in chapter 4, concluding that the concept is misleading. In my view, the almost unquestioned acceptance that most tokens of pain are causally related to physiological activity in a sensory system that is specific to noxious stimuli goes a long way towards explaining why no existing account can address all of P1-P6.

1.2.1 Pain is both perceptual and motivational in nature – P1

significant explanatory problem (see chapter 5). To emphasise my point, *collectively* P1-P6 are a problem because they are not obviously amenable to any existing explanatory account.

The claim that pains *appear* to be constituted by unpleasant phenomenology that varies in quality, intensity and bodily location should not be controversial. These features are evident from introspection of our pains. They are expressed in reports like, "The slight burning pain in the back of my right knee's beginning to really irritate me" and "The stabbing pain in my back is really excruciating".

The variety of the phenomenal qualities of pain, as well as their variable intensity and bodily location are suggestive of perceptual experiences like vision and audition. Moreover, pain reports like those in the preceding paragraph bear a passing resemblance to perceptual reports like "I see there's a blue car parked outside the house" and "I can hear the washing-machine's still going". Pain also draws our attention to a particular part of the body, the place where the pain seems to be located, and we often believe we have tissue damage at that location.

The unpleasantness of pain is also reflected in these reports; the knee pain is *irritating* its subject and the back pain is *excruciating*. It just seems obvious that pain is also affective. We are averse to the unpleasantness of pain, tend to modify our behaviour in ways that ameliorate or eliminate the experience and if our pain does not ease we may suffer as a consequence. There seems to be a necessary connection between pain and unpleasantness or to put it another way, between pain and negative affect. By contrast, I might like the sound of a Beethoven string quartet or the view of the sea, but I might also be indifferent to or dislike either or both. On introspection, our auditory and visual experiences are unlike pain because they seem dissociable from particular affective mental states. There seems to be a contingent relationship between

these perceptual experiences and affect. The asymmetry between pain and these other experiences needs explaining. Why is it that the phenomenal qualities that constitute pain appear to have intrinsic perceptual features like the affectively neutral phenomenology that characterises perceptual experiences and yet they also seem to have an intrinsic affective (motivational) property that perceptual experiences lack?[12]

1.2.2 Pains have dissociable components – P2

Subjects who have had pain treated with morphine, lobotomised patients, and those with a rare syndrome called 'pain asymbolia' ('asymbolia' for short) identify experiences as pain, but report that these experiences are not unpleasant. If pains were *unitary* experiences constituted by characteristically unpleasant phenomenology, these reports could only be explained by calling into question concept possession, application or belief. In short, either these subjects are not deploying *REC* or they are mistaken about the subjective character of these experiences. Certainly, there are reasons to be sceptical about the testimonies of these subjects (pain asymbolia and lobotomies involve brain damage and morphine is an opioid) but on balance at least

[12] One reason to be wary of claiming that pain *is* a phenomenal quality is that it would be a counterexample to standard accounts of phenomenal qualities, which are irreducible *affectively neutral* "properties of mental states that type those states by what it is like to have them" (Chalmers, 1996, p.359, note 2). Chalmers is characterising qualia here, but he equates qualia with phenomenal qualities (1996, pp.3-6). My use of 'phenomenal quality(ies)' should be interpreted this way throughout this thesis. My use of 'phenomenology' is broader. It embraces phenomenal qualities and affect. So a subject's pleasant experience of tasting chocolate and her unpleasant experience of tasting chocolate (because she has eaten too much chocolate) are constituted by different tokens of the same phenomenal quality, but they have different phenomenology; i.e. what it is like to have the pleasant experience of tasting chocolate differs from what it is like to have the unpleasant experience of tasting chocolate.

some of these reports should be taken at face value. [13] Despite appearances to the contrary, there is persuasive evidence that pains are not unitary experiences.

Any lingering doubts about the veracity of these cases should be assuaged by psychological evidence that the intensity of the phenomenology of pains and the intensity of the unpleasantness of pain vary independently of one another (Price, 2000). If pain were a *unitary* experience constituted by unpleasant phenomenology it would be very difficult indeed to explain this psychological evidence without calling into question methodologies. And this strategy would not explain away dissociation syndromes like asymbolia.

The evidence that a pain is a composite experiences constituted by a token phenomenal quality and unpleasantness provides a neat solution to P1; the reason why pain seems to have subjective features characteristic of perceptual and motivational mental states is that pains are actually constituted by perceptual and motivational mental states. Although this solves one problem, it presents a significant obstacle to philosophers who claim that pain fulfils a unitary function. Perceptualists like David Bain and Michael Tye have the problem of explaining the apparently motivational nature of one of the mental states that constitute typical pains, while motivationalists like Colin Klein and myself face the challenge of explaining the apparently perceptual features of the other constituent.[14]

[13] David Bain (2014), Nikola Grahek (2007), Richard Hall (1989) and Colin Klein (forthcoming) all agree.

[14] More specifically Bain and Tye are representationalists and Klein is an imperativist. The specific details of these positions is not of concern to my thesis. My motivationalism differs substantially from Klein's position.

Putting these difficulties to one side, dissociation syndromes like asymbolia require explanation. *What* are those with asymbolia identifying as pain and *how* are they making that identification?[15] There is an obvious answer to the '*what*' question. *Ex hypothesi* those with asymbolia hold and are able to deploy *REC* normally. Devoid of their normal affect the true nature of one of the components of the typical experiences we identify as pain reveals itself as an affectively neutral phenomenal quality that is characteristic of pain. The evidence provided by asymbolia is that one of the components of pain is like the phenomenal qualities of perceptual experiences. Given the careful design of some experiments, a tentative answer to the *how* question is available. It is likely that *in the experimental setting* asymbolics are able to identify a phenomenal quality as a pain because it is sufficiently like the subjective (felt) quality of their pre-morbid (pre-brain damage) pains. Now it is very tempting to generalise from these explanations of '*what*' and '*how*', that we are able to identify experiences as pain because pains have a characteristic phenomenology that individuates all pains (in my terms we recognise the '*proprietary phenomenology*' of pain[16]). In support of this generalisation, I for one have a powerful intuition that my pains have a distinct feel they do not share with any other type of experience. However, this generalisation is not licensed either by this intuition or by research on a handful of rare cases of asymbolia. First, it is at least questionable whether *all* the identifications of pain made by asymbolics are accomplished because their experiences have a recognisable

[15] I concentrate on asymbolia here. There are two reasons for this: first, very few philosophers have considered the evidence that pain is constituted by distinct mental states, but recently three philosophers have based accounts of pain on asymbolia. These accounts feature significantly in chapter two. Second, I am following Grahek in this matter. He argues that lobotomised patients and those on morphine are arguably less convincing examples of dissociation than asymbolia (2007)

[16] In more detail, 'proprietary phenomenology' refers to the ownership of phenomenology, to phenomenology that characterises a type of experience. To illustrate, we *may* be able to recognise pain because either or both of its components are uniquely for pain, but it might also be that pain is uniquely constituted by components that are individually constituents of other (non-pain) experiences. In both cases, pain would have proprietary phenomenology.

phenomenal character. It may be that a contextual factor features in some identifications of pain:[17] the experience of a phenomenal quality that could not be identified as pain under *REC* unless it was simultaneously accompanied by a sudden incident, banging an elbow against a wall, for example. Second, it is possible that some phenomenal qualities cannot be identified as pain under *REC* unless they are experienced, in conjunction with unpleasantness (i.e. unless they are composite experiences).

In summary of this section, the evidence of a few token experiences generated in the experimental setting is not adequate support for the claim that the affectively neutral phenomenal qualities of pain are uniquely for pain. So the initial task derived from the evidence that pain has a composite structure is to set out the commitments associated with two competing positions, that the phenomenal qualities experienced by those with asymbolia are or are not uniquely for pain. If they are constituted by phenomenology that is proprietary to pain, then generalising from the '*what*' and '*how*' of a small proportion of asymbolia pains (the asymbolia pains that have been generated in the experimental setting) is a straightforward matter. It would also support claims that those with asymbolia are experiencing pain. But if they lack proprietary phenomenology, then the explanatory problems mount. I explore these possibilities in the next section.

1.2.3 Characterising the dissociable components of pain – P2

[17] I believe that some "pains" are identified in this way. See '*C-pain*' in sub-section *1.2.3* below, and chapter 6, section *6.1.2*.

The powerful intuition that the experiences we identify as pain are united by proprietary phenomenology suggests a subjective characterisation based on *REC*.[18]

T Pains are the experiences that could be identified as pain under *REC*.[19]

Although I have cautioned against assuming an identity like this it represents a reasonable way of anchoring the term 'pain' for the analysis I will conduct in this sub-section. If the affectively neutral phenomenology of pain is proprietary to pain, then we have a ready explanation for our ability to identify experiences as pain; all the experiences with this phenomenology are identical to all the experiences that *could in principle* be identified as pain under *REC*. This is because successful application of *REC* implies an (in principle) ability to pick out the experiences with this and only this type of phenomenology.[20] In other words, our ability to individuate experiences under *REC* is explained by our recognition of the distinct character of the phenomenology that the affectively neutral component contributes to pains. So the ability to experience pain requires nothing over and above the ability to have an experience constituted by a token of a particular type of phenomenal quality. We would also have a principled means of uniting token experiences under the type pain;

[18] It is worth emphasising that a subjective characterisation of pain is consistent with the IASP's taxonomy (see fn.9). Their recommendation that "if [patients] regard their experience as pain… it should be accepted as pain" (IASP, 2014) is clearly aimed at medical practitioners; doctors and the like should take reports of pain at face value. It does not amount to an endorsement of the view that pains *are* the experiences we could identify as pain under *REC* (*T* above).

[19] To all intents and purposes, Bain (2013), Grahek (2007), Hall (1989) and Klein (forthcoming) endorse *T*. Each takes abnormal experiences that are identified as pain by their subjects as pains (presumably because these subjects are taken to be deploying *REC* or something like it, appropriately).

[20] I say "in principle", because I do not want to make the holding of *REC* a condition on the ability to experience pain. Without this qualification neonates and non-human animals would be incapable of feeling pain. Henceforth, I will not explicitly refer to the *in principle* qualification; readers should take 'could' or 'could be' to imply this qualification.

a token experience is a pain if and only if it is constituted by a phenomenal quality drawn from the quality space that is unique to pain.

The problems associated with this position are strange rather than difficult. One consequence would be that pains are not intrinsically unpleasant; the typical experiences we could identify as pain ('*TYP*' for short) are constituted by pain (a phenomenal quality that types – or characterises – an experience as a pain)[21] and a (non-pain) conscious mental state that makes it feel *as if* the pain is intrinsically unpleasant. This consequence may provide support for perceptualism about pain because the subjective nature of pain would be exhausted by affectively neutral phenomenology that is in turn exhausted by features (varied qualities, intensities and locations) that seem perceptual in nature. The problem with this sharply circumscribed approach would be that it fails to explain why pains are *typically* experienced as if they were intrinsically unpleasant. The problem for motivationalism would be rather different. The aspect of the typical phenomena that we label as 'pain' which is most obviously open to interpretation in motivational terms is not even a constituent of pain. Instead, the motivationalist is obliged to provide a motivational account of characteristic phenomenal qualities (pain) that do not appear to be sufficient for motivation. The pain/non-pain constitution of *TYP* would also have interesting consequences for the standard explanatory model of pain science.

[21] Grahek refers to experiences of these phenomenal qualities in the absence of unpleasant affect as experiences of "pure pains".

By the 'multidimensional model' pains have sensory-discriminative and affective-motivational components (call these components of pain *SC* and *AC* respectively).[22] Some theorists add a further cognitive-evaluative component, but this would be an unnecessary complication; a two-dimensional understanding of this model is sufficient for present purposes.[23] Now it is not entirely clear whether scientists would endorse the view that *SC* and *AC* respectively correspond to the affectively neutral phenomenology and unpleasantness of the composite experiences we typically identify as pain, but my view is that the multidimensional model should be interpreted in this fashion.[24] On this reading the phenomenal qualities have a *sensory-discriminative* function and the unpleasantness has an *affective-motivational* function. As the hypothesis being explored here is that pains are exhausted by these phenomenal qualities, pains are exhausted by *SC*. The implication of this is that the mental state that scientists identify as the affective-motivational *component of pain* is not a component of pain at all. Although these are strange consequences they are relatively trivial, requiring some changes in terminology and conceptual revision, but little more.

[22] The majority of pain scientists accept this conceptual model and it is often cited by philosophers (See Chapman and Nakamura, 1999; Fields, 1999; Tracey and Mantyh, 2007; and Ianetti and Mouraux, 2011. See Aydede, 2000, 2005; Hall, 1989; and Nelkin, 1994 for philosophical discussion of the distinction between pain sensation and affect.)

[23] In an article which traces his development of the concept of a 'neuromatrix', Melzack relates pain (conceived as a conscious experience) to activity in brain areas with sensory-discriminative, affective-motivational, and cognitive-evaluative functions (1999). It is certainly clear from his article and from evidential sources that cognition has the capacity to affect the sensory and affective dimensions of pain and *vice versa*, but it is far from clear whether 'cognitive-evaluative' should be taken to refer to a dissociable component of the subjective experience of pain. I discuss the multidimensional model of pain in chapter 3, section *3.1*.

[24] See chapter 3 for more on this.

At this point, the neutrality and clarity of my discussion will be helped by the use of two placeholders '*Q*' and '*U*' for the constituents of *TYP*.[25]

Q- A placeholder for the phenomenology that subserves the affectively neutral component of *TYP*. My use of *Q* throughout this thesis should not be taken to mean that tokens of *Q* are unique to pain. For all that has been argued thus far experiences other than pain *may* be (partly) constituted by tokens of *Q*.

U- A placeholder for the mental state that makes it feel as if a token of *Q* is intrinsically unpleasant. My use of *U* throughout this thesis should not be taken to mean that *U* is sufficient for the relevant unpleasantness. For all that has been argued thus far it *may* be that a token of *Q* and/or some other mental state is necessary for the relevant unpleasantness.

So using these placeholders asymbolia pain and typical pain experiences can be characterised neutrally:

A-pain An atypical experience constituted by a token of *Q*, which could be identified as pain under *REC*.

[25] The distinction between the typical experiences we identify as pain (*TYP*) and pain is important, because it is an open question whether *TYP* is identical to pain or whether *TYP* is partly constituted by pain (if the affectively neutral phenomenology that partly constitutes *TYP* is sufficient for pain).

TYP A typical experience constituted by a token of *Q* and *U*, which could be identified as pain under *REC*.

If *Q* is constituted by phenomenology that is uniquely for pain, then there is a good reason to accept the following:

1a) An *A-pain* is identical to a pain; i.e. a token of *Q* is necessary and sufficient for pain.

1b) Given 1a and my remarks on the multidimensional model of pain science,[26] pain has a specific sensory-discriminative function.[27]

2a) Given 1a, *TYP* is constituted by pain and a non-pain mental state (*U*) that is not necessary for pain.

2b) Given 2a and my remarks on the multidimensional model of pain science, the affective-motivational function of *TYP* is not a function of pain.

Additionally, there is a robust link between our recognitional abilities and an experience with a specific functional role. An experience of a token of *Q* is apt for its specific sensory-discriminative function because of its intrinsic nature. So pain would be open to definition in terms of this function.

Further details of the hypothetical position that tokens of *Q* are identical to pain would need working out. For example, the fact that we typically experience *Q* as a

[26] See above, this section.
[27] Note that this is not an endorsement of perceptualism as I have characterised it. This is the position that *Q* is sensory-discriminative, which is consistent with both perceptualism and the mixed theory, because perceptualism is the position that both *Q* and *U* have perceptual functions.

constituent of a composite mental state (*TYP*) would need explaining, but this seems a relatively straightforward problem. The specifics of sensory discrimination[28] and the intrinsic subjective nature of the unpleasant affect are more formidable explanatory challenges, but they also challenge the hypothetical position that tokens of *Q* are not uniquely for pain. These issues are considered in detail in coming chapters.[29]

I want to stress that I am only juggling with one ball here: that the phenomenology of *Q* is or is not unique to pain. All other variables are fixed. It is also important to remember that I am working with the understanding of pain expressed in *T*. *If* the phenomenology of *Q* is not unique to pain, then the task of explaining both the nature of *Q* and how those with asymbolia identify tokens of *Q* as pain is much more difficult.[30] By way of example, it is reasonable to assume that in the experimental setting asymbolics identify tokens of *Q* as pain because they are sufficiently like the qualities of their pre-morbid pains (this is implied by the hypothesis that they are deploying *REC*). But if the phenomenology of *Q* is not unique to pain, then tokens of *Q* constitute or are constituents of experiences other than pain, so it seems very likely that only a proportion of tokens of *Q* could be indentified as pain under *REC*. And given *T*, only those tokens of *Q* that could be identified as pain would be sufficient for pain. This raises a question: why could some tokens of *Q* be identified as pain while other tokens of *Q* could not be identified as pain?

[28] The specification of the perceptual object is a particular problem. (In particular, see chapter 2, section *2.1.*)
[29] These explanatory issues are central to my thesis and so they feature in all the coming chapters. But I account for the subjective nature of *Q* in chapter 5, section *5.1.1*, the subjective nature of *U* in chapter 5, section *5.4* and I argue that an experience constituted by a token of *Q* is not sufficient for pain in chapter 5, section *5.3*.
[30] I address this issue in chapter 6, section *6.1.2*.

As those with asymbolia identify experiences constituted by tokens of *Q* as pain, if the phenomenology of *Q* is not unique to pain it is not clear what links the experiences we identify as pain other than our tendency to recognise experiences as such. Consequently, it would seem quite likely that 'pain' labels a motley collection of experiences:

*A-pain** An experience constituted by a token of *Q* that is sufficient for an identification of pain under *REC*.

*TYP** (*i*) An experience constituted by a token of *Q* and *U*, which could be identified as pain under *REC*, where the token of *Q* would be sufficient for an identification of pain under *REC* in the absence of *U*.
(*ii*) An experience constituted by a token of *Q* and *U*, which could be identified as pain under *REC*, where the token of *Q* and *U* would be jointly necessary and sufficient for an identification of pain under *REC*.

To complicate matters these characterisations are not complete. It is quite possible that another factor, a cognitive factor linked to something contextual say, is necessary for an identification of some token experiences as pain under *REC*.[31] In which case:

C-pain: (*i*) An experience constituted by a token of *Q* that is not sufficient for an identification of pain under *REC* and a contextual factor ('*C*'),

[31] I mentioned this possibility by reference to asymbolia in section *2.2*.

where this token of *Q* and *C* would be jointly necessary and sufficient for an identification of pain under *REC*.

(*ii*) An experience constituted by a token of *Q* and *U* that are not jointly sufficient for an identification of pain under *REC* and *C*, where this token of a composite experience (constituted by *Q* and *U*) and *C* would be jointly necessary and sufficient for an identification of pain under *REC*.[32]

These create several interesting issues about the function of pain:

3) Some pains – *A-pains** – have only a sensory-discriminative component.

4) Some pains – *TYP**(*ii*) – have sensory-discriminative and affective-motivational components.

5) It is not clear whether by *TYP**(i), pain has one component (the sensory-discriminative) or both.

6) It is difficult to know how to conceive the contextual "component" of *C-pains*. So the functional status of *Q* in *C-pain*(*i*) and *Q* and *U* in *C-pain*(*ii*) is unclear because they are not sufficient for pain.

It is important to emphasise that *T* is a crucial factor in these explanatory problems. In effect, if *T and* the phenomenology of *Q* is not unique to pain, then there are a lot of explanatory difficulties. So there are two ways of avoiding at least some of these difficulties. The first is to provide a convincing argument that the phenomenology of

[32] I am assuming that an experience constituted by neither *U*, nor *C*, nor *U* and *C* could be identified as pain under *REC*

Q is unique to pain; and the second is to deny *T*.[33] The former strategy leaves relatively minor terminological and conceptual problems of the sort I have outlined above. But I want to emphasise that the argument would have to be convincing, the intuition that pains have a unique phenomenal character would not be a substantial enough reason for this purpose. If no such argument is not available then the second way of avoiding these difficulties should seriously considered.

The irony of this strategy is that proprietary phenomenology offers the prospect of definition of pain that is independent of *T*. If we are able to recognise pains because all pains are unitary experiences constituted by a token phenomenal quality drawn from a quality space that is uniquely for pain (i.e. all tokens of *Q* are pains and all tokens of pain are tokens of *Q*), then there is some justification for the claim that pain is the conscious aspect of a sensory modality. Pain can then be defined in terms of this sensory modality. But if the experiences we could recognise as pain lack an objective grounding like this, then it looks as though the only link between these experiences is a tendency for them to occur at times of injury and a learned ability to categorise them as pain that is based on this tendency. In short, pain is constituted by a motley collection of experiences. In this (hypothetical) light, the sort of constitutional and functional difficulties I have expressed above would be hardly surprising. So the problem here is one of finding a non-arbitrary grounding for a definition of pain that is independent of *T*.

[33] We might of course do both, even though it is not necessary to avoid many of these problems.

In summary, the problem posed by the composite structure of *TYP* boils down to the question of whether or not the phenomenology of *Q* uniquely subserves pain. If it does then the explanatory problems are relatively slight. If not, then either there are a large number of difficult explanatory problems to address, the definition of pain requires revision in some unspecified way or it has to be accepted that the category pain is just a motley collection of experiences that we have learned to categorise together.

In chapter 3 I look at the neuroscience that might underpin an argument to the effect that the phenomenology of *Q* is unique to pain, and in chapter 4 I analyse the 'nociceptive system'. The conclusion of this analysis is that peripheral neuroscience does not support the position that the phenomenology of *Q* is unique to pain and in chapter 5 section *5.2*, I argue that this phenomenology is not uniquely for pain. In chapter 6, section *6.1.2*, I argue that the experiences we identify as pain are a motley collection and in chapter 6 section *6.1*, I define pain in terms of the constitution of *TYP*.

In the following sub-section, I look at the problem of conceiving the components of *TYP* in perceptual, motivational or mixed terms so that they fulfil the overall function of pain.

1.2.4 The function of pain – P3

Subjects who are unable to experience *Q* as if it were intrinsically unpleasant in a part or parts of the body because of nerve damage are prone to injuries and secondary infections.[34] This is persuasive evidence that the composite experience constituted by *Q* and *U* functions to prevent injury. Everyday observation of normal subjects reveals at least part of the way in which the composite fulfils this function; simply put, the composite motivates subjects to change behaviour that could be causing damage. Those with asymbolia, who are unable to experience *U*, also suffer a greater number of and more severe injuries than those able to experience the composite. This evidence supports the claim that an experience of *Q* is not sufficient to adequately motivate injury-preventing behaviour and *U* fulfils a necessary motivational function as a constituent of *TYP* in the motivation of injury-preventing behaviour.[35] As *Q* has features of a perceptual mental state it is reasonable, at this stage, to assume that *Q* has perceptual content representing an aspect of the mind-independent world that is relevant to the motivation of injury-preventing behaviour. This assumption is consistent with the *prima facie* view that *Q* is constituted by phenomenal qualities, because on a standard conception phenomenal qualities have no intrinsic affect.[36] The

[34] These subjects have 'peripheral neuropathies'. Amongst many different causes, peripheral neuropathies are symptoms of, for example, diabetes and leprosy. Congenital analgaesia (sometimes called 'congenital insensitivity to pain') is often cited as a case of a global inability to experience pain, but the analysis above raises doubt about what this means. Given *T*, either all pains are constituted by *Q* or some pains are constituted by *Q* and the remainder are constituted by *Q* and *U* (ignoring '*C-pains*'). On both alternatives an inability to experience pain implies an inability to experience the tokens of *Q* that could be identified as pain under *REC*. Because some subjects with congenital analgaesia report feeling strong mechanical sensations it is at least possible that these are experiences of *Q* that could be identified as pain under *REC*. If this is correct, it may well be that *these subjects* fail to identify these experiences as pain because they do not hold *REC* (because they are born with congenital analgaesia). So congenital analgaesia can be explained as an inability to experience *Q* and/or *U*, or as an inability to put *Q* and *U* together to form the composite experience that we normally identify as pain. Because of this uncertainty I have not used congenital analgaesia as an example in the main text. (For more on congenital analgaesia see Melzack and Wall, 1996 p.p.3-7.)

[35] To say that pain is involved in injury prevention is a gross oversimplification but it is enough here, so I am using the term 'injury-preventing behaviour' for convenience. I discuss the function of *TYP* in more detail in chapter 5, section *5.5*.

[36] See fn.12, this chapter.

question is what this content might be, and how do the perceptual content of *Q* and the affect of *U* combine to motivate injury preventing behaviour.

The views of philosophers like Armstrong (1962), Bain (2012, 2014), Hill (2009), Grahek (2007), Pitcher (1970) and Tye (2005a, b) amount to the claim that the perceptual function of *Q* as a constituent of *TYP* is to represent tissue damage. On a conservative view of this content, *Q* represents the location, intensity and nature of tissue damage.[37] For example, an experience constituted by a moderately intense burning quality which is felt as if it is located in the back of the right hand represents a moderate burn on the back of the subject's right hand. The problem is that it is far from obvious what role this content might play in motivating injury-preventing behaviour.

A visual experience representing a moderate burn on the back of a subject's right hand is likely to be a crucial factor in that subject judging that she has a burn on the back of her hand. In turn, this judgement would be a factor in the triggering of a mental state motivating her to put a sticking plaster on the burn. But she might equally decide that healing would be better served if the burn is left uncovered or she might not care at all about the burn. With respect to vision, perceptual content and motivation are most often linked by judgements, beliefs and so on. As such the relationship between perceptual states and motivation is extremely variable. This is not the case with *TYP*. I do not need to make a judgement about the nature, severity

[37] Richard Hall takes a liberal view of this content. He claims it represents "whether the bodily damage is merely imminent, is actually occurring, or is left over from past wounds; what its location is; what type of damage it is (burn, cut, bruise, etc.); and how bad it is (Hall 1989, pp.644). I discuss the specifics of the views of these philosophers in chapter 2, section *2.1.1*.

and location of tissue damage and an appropriate course of action to be motivated by *TYP*. There is another important asymmetry between vision and *TYP*. If a visual experience leads a subject to believe she has a moderate burn on the back of her hand but she is then convinced she is hallucinating, she is unlikely to be motivated to put a plaster on her injury. By contrast, someone experiencing *TYP* with content that is misrepresenting the state of her right hand would be motivated by her experience even if she believed she had not damaged her hand. The most natural explanation for this asymmetry is that unlike vision, when a subject is experiencing *TYP* she is motivated by the feeling of *TYP* itself and not by perceptual content.[38] As this content plays no obvious functional role in *TYP* perceptualism about pain is in doubt.

The evidence that *U* fulfils a necessary function in a composite mental state that motivates injury-preventing behaviour is an important strand in motivationalism about pain, but motivationalists face the opposite content-related problem, how to cash out their position without ascribing perceptual content to *Q*. I have been discussing motivation by referring to '*injury*-preventing behaviour'. On these terms, motivation seems to require injury-representing content. The worry is that viable versions of both perceptualism and motivationalism are going to look like versions of the mixed theory; i.e. each would be a mixed theory. However, despite the advantage of not being obliged to explain both *Q* and *U* in the same functional terms, mixed theorists face a problem of detail, the explanation of how (on their account) the perceptual content of *Q* functions in concert with the motivational function of *U* to yield injury-preventing behaviour.

[38] Murat Aydede makes a similar point in his 2009.

My intention in this section has been to present some challenges facing theorists of all stripes. It has not been my intention to analyse the merits of existing theoretical positions with respect to the function of *Q* and *U* in *TYP* or to present possible solutions to these problems. The relationship between the functions of *Q* and *U* requires explanation because of the gestalt-like constitution of *TYP*. These functional issues are problematic for perceptualists, motivationalists and mixed theorists alike.

I discuss specific ways in which perceptualists, motivationalists and mixed theorists might address these functional/constitutional issues in chapter 2, sections *2.1.5*, *2.2* and *2.3* respectively and present my motivational solution in chapter 5, section *5.6.2*.[39]

1.2.5 The weak correlation between pain and the stimulus – P4 -P6

The 'pain' in the title of this sub-section refers to the typical experiences we recognise as pain (*TYP*). I have divided the evidence of a weak correlation between *TYP* and the stimulus[40] into three categories; the evidence that *TYP* is often experienced in the absence of the stimulus (P4), the evidence that the stimulus often occurs in the absence of *TYP* (P5), and the evidence that the intensity of *TYP* does not correlate well with the intensity of the stimulus (P6). Before discussing this evidence, I want to mention three caveats that apply in this section.

[39] Scientists conceive the constituents of *TYP* as sensory-discriminative and affective-motivational components, but they do not address the detail of how these functions yield injury-preventing behaviour. In chapter 3 section *3.4*, I interpret the conceptual models of pain science in a way that is consistent with the mixed theory I set out in chapter 2 section *2.3*.
[40] This is widely observed. For an overview, see Moseley (2007).

First, although perceptualism about *TYP* ('perceptualism' for short) does not carry a commitment to the position that tissue damage is the perceptual object, throughout this section I take perceptualism to be the thesis that *TYP* represents *tissue damage*. I do this because most philosophers of pain, whether perceptualists or not, assume that if pain has a perceptual object then tissue damage is that object.[41] So conceived P4-P6 present a very significant obstacle to perceptualism. One way of at least partly addressing these problems is to discard this assumption in favour of a different perceptual object, noxious intensities of energy, but I will reserve full discussion of this revision for chapter 2, section *2.1.2*.

Second, and in a similar vein, I take it that if tissue damage is the perceptual object it is also the stimulus. Putting this second caveat together with the first, the implication is that tissue damage is the perceptual object and the stimulus (it is detected by the relevant sensory receptors). This strategy simplifies matters because some of the evidence I will present refers explicitly to the stimulus while some concerns tissue damage. Of course, perceptualism does not entail the view that the stimulus is the perceptual object. Superficial consideration of visual experience illustrates this perfectly. The photoreceptors in the retina of the eye are stimulated by light, but to suggest that visual perception is the perception of the stimulus would be off the mark. Our ability to see mind-independent objects (or their properties) and their spatial relations rather than the light can be explained in evolutionary terms that I will not discuss here. The relevant issue is that the properties of objects, light and

[41] For example, David Armstrong writes of pains being sense-impressions of 'bodily or physical disturbances' (1962, p.p.108-109), Norton Nelkin of pain representing 'harmful states of the body'(1994, p.332), and Michael Tye of pains tracking 'tissue damage' (2005a, p.101).

photoreceptors are links in the causal chain that leads to visual phenomenology. If this were not the case the connection between the perceptual object and visual experiences would be mysterious and philosophically troubling as a consequence. In the context of *TYP*, if tissue damage is not the stimulus, it is a very close link in the relevant causal chain. In taking perceptualism to be the view that tissue damage is the stimulus, I am simply saying that tissue damage can be treated as such for the sake of the argument.

Third, the analysis in preceding sub-sections raises questions about the reports that form the basis of the conclusion that *TYP* is only weakly correlated with the stimulus. In particular, reports underpinning the conclusions expressed in P5 and P6 are open to doubt. With respect to cases where severe injury occurs but the subject denies feeling *TYP* (in sport, for example) it is plausible that she experienced a token of *Q* that could be identified as pain under *REC*, but did not identify her experience as such because she did not notice the nature of her phenomenal experience. This is explained by the absence of unpleasant affect (her experience lacked the non-pain component *U*). With respect to P6, it is possible that *Q* closely tracks the severity of tissue damage, but *U* does not. Unless the composite nature of *TYP* is a feature of experimental designs the evidence that the intensity of *TYP* does not correlate well with the stimulus should be treated with caution. It might be that *Q* correlates reasonably closely with the stimulus in both P5 and P6. In this sub-section, I will take the evidence for P4-P6 as evidence of a weak correlation between the stimulus and *TYP*, not as evidence of a weak correlation between *Q* and the stimulus.

Melzack and Wall cite chronic pain syndromes like some low back or neck pains, tension headaches and trigeminal neuralgia[42] as examples of *TYP* in the absence of tissue damage (1996 p.9), but cases like this are far more commonplace than these examples might lead us to believe.[43] As I sit here, I can feel a vague aching in my buttocks. If I were to stretch my forefinger back into extension or pull the hair on the back of my hand I would feel *TYP*. Experiences like this are part of everyday life. The trouble is that it is far from clear whether these incidents involve tissue damage. If they do not involve tissue damage, then a high proportion of all *TYP*s are misrepresentations and perceptualism is in serious difficulty.

The IASP's definition of pain suggests a response to this problem; pain is "An unpleasant sensory and emotional experience associated with actual or potential tissue damage…" (2014). The defence being that the sitting, stretching and pulling mentioned above are 'potential tissue damage'.[44] Unfortunately, potential tissue damage is a metaphysically dubious concept because it suggests a tissue state. As it would be difficult to specify a tissue state that is neither damaged nor undamaged the most coherent (and charitable) understanding of 'potential tissue damage' is that it is an *event* involving an intensity of energy that has the potential to damage tissue. This

[42] Severe pain in response to innocuous stimulation (like light touch) in the distribution of the trigeminal nerve (the face).

[43] Murat Aydede writes: "Chronic pain syndromes are not restricted to rare cases like phantom limb pains and referred pains (although the latter are more common than the former). There are more than 1500 pain clinics in the US alone mostly devoted to treating chronic pains, the majority of which are centrally caused pains felt in bodily locations that are not in any pathological conditions. 40% of all Americans suffer from chronic pain at least once and usually late in their lives" (Aydede 2009, pp.537 footnote 7). However, it is far from clear whether this a worry for the representationalists that are the target of his argument because the question is one of proportion; what proportion of *all* pain experiences are chronic pains of this type?

[44] This defence is clearly available to Hill who writes of his 'perceptual/somatic theory' that the experiences we identify as pain are representations of "bodily disturbances involving actual or potential damage" (2009, p.189).

interpretation is consistent with the position that pain has content representing noxious intensities of energy (see the first caveat above). I put this matter to one side for now but it features heavily in the revision to perceptualism I propose in chapter 2, section *2.1.2*.

The observation that tissue damage is often present in the absence of pain poses a further problem. The most obvious examples come from sport and war, but Ronald Reagan denied feeling pain when he was shot on March 30th, 1981 (Wall, 2000). In such cases, subjects often report feeling a mechanical sensation (like a thump, snap or crack) but deny experiencing pain.[45] Experiences like these illustrate the sort of explanatory concerns I expressed in the third caveat above. They may be experiences of *Q* that could be identified as pain under *REC*, but their subjects did not attend to them sufficiently closely *at the time they were experienced*. Retrospectively, their recognitional abilities let them down because they do not remember their experiences sufficiently accurately to identify them as pain under *REC*. The absence of an experience of *U* explains the inattention and inaccuracy of their memories. Alternatively, they may be experiences of *Q* that could not be identified as pain under *REC* (if *Q* is not specific to pain)[46] or they may be experiences constituted by phenomenal qualities other than *Q* (phenomenal qualities that could not be identified as pain under *REC* in any circumstances even if they were constituents of experiences partly constituted by *U* and/or *C*). These possibilities give perceptualists and mixed theorists some wiggle room, as all that we can be (more or less) sure of is these are cases in which tissue damage occurs in the absence of *TYP*.

[45] This evidence comes from personal experience; I worked as a physiotherapist for twenty years. See also Melzack and Wall (1996) and Wall (2000).
[46] See *TYP**(*ii*), *C-pain*(*i*) and *C-pain*(*ii*) and the discussion in sub-section *1.2.3*, above.

Scientists explain cases like this these in terms of context; *TYP* is inhibited because it has the capacity to distract subjects whatever their circumstances. In certain contexts this could compromise survival. Of course, most sports do not literally involve survival, but it is easy to see that inhibitory mechanisms which have evolved for the purpose of survival may well be factors that influence pain in sporting situations. The Reagan case is not obviously amenable to this sort of explanation. It may be that cases like this are rarities. If so they can be dismissed as anomalies; as rare failures to represent tissue damage. But this may not be the case. I am not aware of any scientific or philosophical accounts that attach any particular importance to cases like this. It seems that all cases of the absence of *TYP* when frank tissue damage occurs in circumstances of sport and war are treated in the same way; they are treated as cases of physiological inhibition of sensory input. I provide reasons to doubt this explanation in chapter 6, section *6.1.2*. In my view, this is a commonplace occurrence. If this is right it may be that some cases of the occurrence of severe tissue damage in the absence of *TYP* in circumstances of sport or war are also not cases of inhibition.

More problematically, it is not commonly recognised that tissue damage is *most often* present in the absence of *TYP*. When we have tissue damage we are most often *TYP*-free. To see this consider that you may well have tissue damage somewhere in your body right now (some arthritis in a joint, a slightly damaged ligament or cut, say) even though you are probably not experiencing *TYP*. Cuts, bruises, sprains, strains, fractures and so on take time to resolve. It would be very unusual if we were to experience *TYP* during the whole course of tissue resolution. If we did experience

constant *TYP* it would be a symptom of something other than the tissue damage, an infection perhaps. In the vast majority of cases *TYP* is episodic. We are likely to experience *TYP* when we twist an ankle, but that *TYP* will disappear relatively quickly (in a matter of minutes or hours, in contrast to the weeks it will take the tissue damage to resolve). To be sure, putting weight on the ankle is likely to cause *TYP* in the early stages of recovery, but that *TYP* will also disappear if weight is taken off the ankle. If *TYP* did represent tissue damage these would be failures to represent rather than misrepresentations, but this would not mean they could be ignored by perceptualists. If the majority of visual experiences failed to represent it would be a catastrophe; if we continued to rely on vision we might fail to survive so it seems likely we would disregard our visual experiences.[47] In which case, it would be hard to explain our ability to experience visually in evolutionary terms as visual experience would not seem to confer any advantage on its subjects. The claim that *TYP* represents tissue damage should be amenable to this sort of explanation. As the evidence is that *TYP* is *grossly* inaccurate as a representation of tissue damage there is no plausible evolutionary explanation for our ability to experience *TYP*. I discuss these difficulties in detail in chapter 2, sections *2.1.1* and *2.1.2*.

Finally in this section, even when pain is correlated with the stimulus, the evidence is that there is not a one to one relationship between the intensity of the stimulus and the intensity of *TYP* (Moseley 2007, p170)). This experimental evidence is supported by

[47] It might be responded that this is not analogous to *TYP*. The failure to visually represent a bus might well be a factor in a fatal accident, but this would be an hallucination; the subject is hallucinating the background features that would otherwise be obscured by the bus. I can concede this point, but it does not dilute the problem. Evolution implies advantage. It is implausible to claim that our ability to experience *TYP* has not been evolved so *TYP* must have conferred an advantage to our ancestors. If the ability to experience *TYP* has evolved to represent tissue damage and it most often fails to represent tissue damage, what might that advantage be?

everyday observation. Consider an individual with a sprained ankle who is *TYP*-free when she is sitting down:[48]

Sprain A subject with a sprained left ankle has been sitting watching television for a couple of hours. She is *TYP*-free even though her ligaments are sprained. She rises from her chair. In so doing, she puts a very small amount of body weight on her left foot and experiences quite intense *TYP* as a consequence. Nevertheless, she carries on and begins to limp around the room. The continued movement is accompanied by a steady decrease in the intensity of her *TYP* and so she slowly increases the intensity of the weight-bearing force on her ankle. After six minutes or so her *TYP* has eased to mild discomfort despite the fact that she is taking most of her body weight on her left foot as she walks. Soon she sits down again, but almost immediately the telephone rings and she stands again. This time her ankle *TYP* is very slight.[49]

This is an example in which the subject's tissue damage is constant but she experiences a very significant change in the intensity of *TYP*. A little reflection on the variation in the intensity of *TYP* you have experienced when you have been injured should be enough to convince that cases like this are commonplace.[50] As such they represent a considerable (perhaps the most significant) empirical challenge for any explanatory account of pain. In chapter 2, sections *2.1*, *2.2* and *2.3* and chapter 3,

[48] This itself is a problem for the perceptualist. (See cases of tissue damage in the absence of *TYP*; this sub-section above)
[49] Cases like *Sprain* are a significant challenge to all explanatory accounts of *TYP* so I repeat this case in chapter 2, section *2.1.3* and in chapter 6, section *6.3.3*.
[50] My own experiences have been much like this, but this case is drawn from my work as a physiotherapist. (See also fn.45 above.)

section *3.4* I argue that perceptualism, motivationalism, mixed perceptualism and the scientific models of pain respectively cannot adequately explain cases like this. I account for this specific case in chapter 6, section *6.3.3*.

The evidence I have presented in this sub-section is a challenge for all theorists, but it is a particular problem for perceptualism and mixed perceptualism/motivationalism because *TYP* is *grossly* inaccurate as a perception of tissue damage. There may be some wiggle room for mixed theorists at least because the evidence I have presented concerns *TYP* rather than *Q*, but perceptualism and the mixed theory conceived in terms of tissue damage look to be in serious trouble. The imperativist Colin Klein appears less vulnerable in this respect.

Although imperativism is an appealing way of cashing out motivational drive, imperativism does not equate with motivationalism on my classification. For example, Manolo Martinez and Richard Hall conceive the content of *TYP* in terms that are consistent with mixed perceptualism/motivationalism. According to Martinez *TYP* has the imperative content that "this bodily disturbance is no more" (Martinez 2011, p.78). In other words, the imperative has content representing the nature, severity and location of tissue damage.[51] Hall's position is similar; *TYP* has compound intentional content part representational and part imperative. The former represents the "afflicted area as physically damaged in some way or in imminent danger of damage" (2008, p.534). Both of these versions of imperativism share the problems of existing perceptualist accounts, they specify content in terms of tissue damage, so they face the

[51] Martinez might deny he needs this sort of detail. The location and intensity of the damage would do. However, this would not dilute the problem.

challenge posed by the weakness of the correlation between tissue damage and *TYP* (P4-P6). The imperativist Colin Klein is less easily pigeonholed. On the face of it his position is not obviously at odds with P4-P6 because he does not express imperative content in terms of tissue damage. On his account *TYP* is a negative imperative with varied and complex content that can be summed up as, 'stop doing some particular action that is potentially harmful to a specific body part' (Klein, 2007). Whether or not this content qualifies him as a motivationalist is debatable, but I have interpreted his recent (as yet unpublished) account in terms of motivationalism.[52]

Klein is in a stronger position than imperativists like Martinez and Hall, mixed theorists and perceptualists, because potentially harmful action can be construed in terms of potentially harmful intensities of energy. The idea being that inappropriate action imposes a demand that exceeds a tissue's ability to cope. This position can explain many of the problem cases expressed in this sub-section. When I am sitting, stretching or pulling I am subjecting my body to intensities of energy that have the potential to cause damage. This strategy also explains the commonplace experience that *TYP* is most often absent in the presence of tissue damage (P5). Klein's position accurately predicts what actually happens in such cases; subjects experience *TYP* when they act in ways that impose too much force on the damaged tissue. So drawing on Klein's position we can revise perceptualism in a helpful way; *TYP* represents noxious intensities of energy. In chapter 2, I discuss these issues in depth and revise perceptualism accordingly.

[52] See chapter 2, section *2.2*.

In summary of this chapter, I have identified the *explanandum* of my thesis as the experience that we identify with the word 'pain' in everyday thought and talk. Broadly speaking, three existing theories provide competing explanations of this experience: perceptualism, motivationalism and mixed perceptualism/motivationalism. In order to decide which provides the best account of pain I have presented six explanatory challenges. The best theory is the one that provides the best explanation for *all* these problems. If no existing theory has the capacity to explain all six problems then none is an adequate theory of pain.

The first of these problems (P1) – the *prima facie* evidence that pains are constituted by characteristically *unpleasant* qualities that vary in intensity and location unlike other phenomenal qualities which are affectively neutral – is solved by the evidence for the second problem (P2). Pain asymbolia provides persuasive evidence that pains are constituted by affectively neutral phenomenal qualities and an affective mental state, but it creates further explanatory problems. These problems are less testing if the phenomenology of the affectively neutral component is unique to pain, but either way an adequate account of the constitution of pain must explain how each component contributes to the overall function of motivating injury-preventing behaviour (P3), and why the correlation between pain and the stimulus is weak (P4-P6).

In chapters 2 and 3, I argue that no existing account can explain all of P1-P6. In chapters 5 and 6, I present near-motivationalism and argue that it is a solution to all six problems.

2 PHILOSOPHICAL ACCOUNTS OF PAIN

In section *1.2.2* of chapter 1, I introduced a syndrome called 'pain asymbolia'. Asymbolia supports the view that the experiences we typically identify as pain are composites constituted by an affectively neutral phenomenal experience ('*Q*') and a further mental state ('*U*'), which makes it feel as if *Q* is intrinsically unpleasant. Asymbolia raises many difficult questions about pain. David Bain, Nikola Grahek, Richard Hall and Colin Klein take the view that we should take pain reports at face value, as reports of pain. As those with asymbolia identify experiences of *Q* in the absence of *U* as pain, the implication of their view is that *U* is not necessary for pain. At this stage, I wish to remain neutral about this matter;[1] I also want to avoid the ambiguous use of 'pain'. So to avoid misunderstanding, I am using '*TYP*' to refer to the composite experiences (constituted by a token of *Q* and *U*) we typically identify as pain or locutions that make it clear whether I am referring to an experience constituted by just a token of *Q* or the composite. Where *quotations* refer to 'pain', they are invariably referring to the composite, *TYP*.

In the introduction to chapter 1, I divided the theoretical territory into two competing camps, perceptualism and motivationalism and a hybrid perceptual/motivational position I call the 'mixed theory' or the 'mixed position'. Even though the detail of the specific philosophical positions that fall into these categories carry significant

[1] I argue that a token of *Q* is not sufficient for pain in chapter 5, section *5.3*.

philosophical commitments (to 'transparency' and 'satisfaction conditions' for example[2]) these broad groupings are intended to be uncontroversial. Perceptualism is the theory that both *Q* and *U* fulfil perceptual functions as constituents of *TYP*, while motivationalism is the theory that both *Q* and *U* fulfil motivational functions as constituents of *TYP*. The mixed theory ascribes a perceptual function to *Q* and a motivational function to *U*.

In this chapter, I will argue that neither perceptualism nor any current motivational account can adequately address the constitutional, functional and empirical problems posed by *TYP*.[3] The mixed theory is more promising because it handles *Q* and *U* in what seems to be a natural way, as perceptual and motivational mental states respectively. Although I raise a couple of concerns about this theory, in particular I question the functional role of perceptual content, these are relatively minor worries when balanced against the explanatory advantages of the mixed theory and its apparent consistency with pain science.

This chapter is divided into three parts. The first part (*2.1*), which focuses on perceptualism about pain, has three sub-sections. In *2.1.1*, I argue that perceptualism conceived as the theory that tissue damage is the perceptual object of pain is fatally flawed. I devote considerable space to this argument because almost without exception perceptualists and mixed theorists take tissue damage to be the perceptual

[2] I do not attack representationalism or imperativism (say) on the basis of philosophical commitments like these. My sole concern is whether accounts like these have the power to explain P1-P6.
[3] I am taking Colin Klein's as yet unpublished account as the only existing example of motivationalism. With respect to my categories, Klein's account is rather undeveloped so it may be that he would object to my reading of his account. If he did object then I am not aware of any existing motivationalists other then myself. Manolo Martinez and Richard Hall who, like Klein, are described as imperativists are mixed theorists on my classification.

object of pain. I argue that the problems presented by P4-P6 and by sensory neurology are overwhelming. In *2.1.2*, I suggest fixing perceptualism (and the mixed position) by identifying the perceptual object with noxious intensities of energy. In *2.1.3*, I question whether the revised version of perceptualism can adequately account for the composite structure of pain, and conclude that it cannot. In section *2.2*, I concentrate on Colin Klein's version of motivationalism arguing that his account is an ad hoc attempt at preserving a unitary theory of pain. In *2.3*, I consider mixed perceptualism/motivationalism, concluding that it has significantly more explanatory power than either perceptualism or Klein's motivationalism.

2.1 PERCEPTUALISM

Perceptualism is the position that *TYP* functions as a representation or an awareness (in the veridical case) of some feature of the mind-independent world. Almost without exception, perceptualists specify tissue damage as the perceptual object of *TYP*. So a token of *TYP* has content about tissue damage. I call this position 'TD perceptualism'. In chapter 1 section *1.2.5*, I presented what I consider to be overwhelming difficulties for this position. I explain why TD perceptualism is unsustainable in sub-section *2.1.2*, but first I want to forestall the claim that my effort is wasted because TD perceptualism is not the dominant perceptual theory of pain.

2.1.1 TD Perceptualism

Some perceptualists specify the perceptual object of *TYP* as 'bodily disorders' (e.g. George Pitcher) or 'bodily disturbances' (e.g. David Armstrong, David Bain and Christopher Hill) without explaining what they mean by these terms. For example, Brian Cutter and Michael Tye see both tissue damage and disturbances as perceptual objects:

> The representational content of pain experience is exhausted by information about the location and physiological properties of some tissue damage or disturbance (2011, p.91).[4]

And George Pitcher thinks tissue damage is a sub-type of bodily disorder:

> To feel pain in a certain part of one's body, according to the perceptual theory, is to feel (that is, perceive) a disordered state of that bodily part – that is, to feel a bodily part that is in a damaged, bruised, irritated or pathological state, or in a state that is dangerously close to being so (1970, pp. 389-390).

It is clear from this that Cutter, Tye and Pitcher are not *identifying* the perceptual object with tissue damage. Still, unlike Armstrong, Bain and Hill, it is also clear they endorse the view that tissue damage is the perceptual object of some *TYP*s.[5] Although

[4] In an earlier account Tye does not qualify matters, "Pain experiences normally track tissue damage" (2005a, p.101).

[5] Armstrong adds a little more substance. For him 'bodily disturbances' are 'bodily *states*' (Armstrong, 1962, p.106). Tissue damage is clearly a bodily state which typically involves anatomical *and* physiological disturbance or disorder. But what might be added to the list of bodily states other than tissue damage is a matter of speculation. Bain's position is unclear. He too equates 'bodily disturbances' with bodily states (2013, p.S71, fn.7) and takes 'aptness to harm' to be an appealing candidate for perceptual object (2013, p.S82, fn.31). However, he does not explain what bodily state

Cutter and Tye are not forthcoming about 'tissue disturbances', Pitcher explicitly states that bruised and irritated bodily tissue along with (some) pathological tissue states are also perceptual objects. Presumably, these other philosophers would agree. If this is right, they are agreeing to a slightly confused division of the pathological territory. Bruising and irritation (I assume Pitcher is referring to inflammatory states here[6]) are part and parcel of the category 'tissue damage' and tissue damage is a pathological state. Perhaps these philosophers would add some disturbed or disordered physiological states that do not involve tissue damage to the list of pathological states that are the perceptual content of pains, but this is conjecture. What matters here is that all these perceptualists would agree that tissue damage (conceived broadly to include frank damage, bruising, some scarring, inflammatory conditions and so forth) is the perceptual object of most veridical *TYP*s. In summary then, the TD perceptualist is not a straw man. TD perceptualism is an (almost) all-pervading thesis and, I will argue, it is harmful for the cause of perceptualism.[7]

2.1.2 The falsity of TD Perceptualism

'aptness to harm' is intended to label. Hill does not explain what he means by 'bodily disturbance' in his 2009, but in his 2005 his bodily disturbances involve tissue damage.

[6] Inflammation is complex. Particular chemicals are released from damaged tissue as well as cells in the vicinity of tissue damage. These chemicals function to prevent infection and aid tissue repair, but they are also involved in the breakdown of tissue so, in a sense, they have a tissue-damaging effect. Unchecked, some of these 'inflammatory mediators' can cause significant tissue damage. These chemicals might be cited as disordered pathological states (see the main text below) distinct from tissue damage, even though they have the capacity to cause tissue damage.

[7] I am equivocating because there is an ambiguity to this matter. As well as the talk of 'bodily disturbances' Nikola Grahek writes of *TYP* representing both 'tissue damage' and 'noxious stimuli' (2007). There is a great deal of difference between these candidates for perceptual object. The former is a *tissue state*, while the latter is an *event* involving intensities of energy. My guess here is that Grahek is taking tissue damage to be a noxious stimulus. If this is right, Grahek conforms to my generalisation.

It surprises me that TD perceptualism is so dominant given that the evidence of *TYP* in the absence of tissue damage (P4) and tissue damage in the absence of *TYP* (P5) is both commonplace and available to armchair reflection. To summarise the evidence I presented in section *1.2.5* of chapter 1, experiencing vague aching in the buttocks as a consequence of sitting for longer periods and the predominant absence of *TYP* during the whole period in which tissue is damaged are everyday examples of P4 and P5 respectively. Anyone tempted to put this evidence down to non-veridical experience (evidence for P4) or perceptual failure (evidence for P5) would be obliged to explain the asymmetry between the accuracy of *TYP* and perceptual experiences like vision. Visual experiences are accurate *enough* to help us to navigate the world. But given the gross inaccuracy of *TYP* it is extremely doubtful whether it could provide any significant advantage as a means of perceiving tissue damage. And if *TYP* could not provide a significant advantage it would have to be explained how it is that we have evolved the capacity for *TYP*. Indeed, it is difficult to explain what advantage might be gained by having experiences that represent tissue damage.

From the evidence provided by those unable to experience *TYP* (e.g. subjects with peripheral neuropathies) or *U* (e.g. subjects with asymbolia), *TYP* fulfils a crucial function, the motivation of injury-preventing behaviour.[8] How might the content moderately burned skin on the lateral aspect of the right foot (say) fulfil this function?[9] To see the functional problem consider that TD perceptualism predicts the following of someone sitting warming his feet at the hearth of an open fire:

[8] See chapter1, section *1.2.4.*
[9] I make a similar point in chapter 1, section *1.2.5.*

Fire As the temperature in the tissues of his right foot slowly rises towards the threshold (47°C) at which tissues start to become damaged (the '*damage threshold*') he would experience an intensifying heat sensation, but no *TYP*.[10] Once the tissue temperature reaches 47°C damage would begin to occur. This would be perceived as a *slight* burning *TYP* in his right foot. As such it would be easily ignored or even pass unnoticed and more extensive damage would occur. This would lead to an intensification of *TYP* and a greater likelihood of him withdrawing his feet from the fire. But the intensity of *TYP* would not change when he changes his behaviour because the severity of the damage would not be altered by the behavioural change. He has sustained damage of severity *x* which is perceived as *TYP* of intensity *3*. As damage of severity *x* would persist for a considerable period after he removes his foot from the hearth so too would *TYP* at intensity *3*. The intensity of *TYP* would only change as the burn begins to heal.

If *Fire* were accurate, it is hard to see the utility of *TYP*. By TD perceptualism, pain would not prevent damage and given the persistence of (a veridical) *TYP* subsequent to injury it is difficult to see what role this persisting *TYP* might play in motivating appropriate behaviours following tissue damage. But crucially *Fire* seriously misrepresents actual cases. In particular, *TYP* would ease when the subject removes his foot from the hearth.[11] On the reasonable assumption (given the evidence of P3) that *TYP* is involved in injury-preventing behaviour, it seems obvious that the

[10] The damage threshold of 47°C comes from Leach, E.H., Peters, R.A., & Rossiter, R.J. (1943) 'Experimental Thermal Burns, Especially the Moderate Temperature Burn', *Experimental Physiology* 32, p.67-86.
[11] For a more realistic account of *TYP* experience, see *Ankle* below.

important perceptual content is not that *I am* damaged in some way to some degree at some location, it is that *I am going to be* damaged or further damaged to some degree at some location.

In chapter 1 section *1.2.5*, I mentioned a possible response to this problem. By Bain's evaluative thesis *TYP* represents tissue damage or the *threat of damage* (2014, p.306; my emphasis) and Hill claims pains are "representations of bodily disturbances involving actual or *potential damage*" (2009, p.187; my emphasis). The idea being that some injuries, like the burn in the illustration above, would be prevented because the subject would perceive the potential for tissue damage before the damage occurred. Despite the fact that this sort of characterisation is consistent with the IASP's definition of pain,[12] the idea that 'potential tissue damage' is a bodily state[13] is problematic. On a straightforward understanding of the conceptual possibilities tissue is either damaged or undamaged. Given that 'potential tissue damage' is often contrasted with 'actual tissue damage', I take it that 'potential tissue damage' does not refer to damaged tissue, so potential tissue damage is undamaged tissue. The problem then is to provide some principle for distinguishing the undamaged tissue that is categorised as 'potential tissue damage' from the undamaged tissue state that is not 'potential tissue damage'. This is particularly difficult because the straightforward understanding that tissue is either damaged or undamaged is a misleading oversimplification. Almost all types of tissue are constantly being regenerated through

[12] The IASP define pain as, "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (2014)

[13] This is an important qualification. If 'potential tissue damage' (or 'threat of damage') is taken to refer to *events* that have the potential to damage tissue, then 'potential tissue damage' makes more sense. The problem would then be that potential tissue damage, conceived as an event, is inconsistent with TD perceptualism, which concerns tissue *states*. To see this consider that tissue damage is a tissue state but it is not an event, and the damaging of tissue by thermal energy is an event but it is not a tissue state. The notion of threat is a complex issue which I discuss in more detail in chapter 6, section *6.2.1*.

processes of *breakdown* and rebuilding.[14] So there is no clear distinction between damaged and undamaged tissue. In a sense then, almost all tissues are constantly damaged. In this light, the prospect of carving the conceptual territory into three meaningful categories that are consistent with biological science is bleak.

The evidence of a weak correlation between the intensities of *TYP* and tissue damage (P6) is even more troubling for TD perceptualism. Here it is not necessary to refer to difficult cases like *Sprain*;[15] straightforward cases, which can be handled by the version of perceptualism I will develop in the coming sub-section, present a seemingly insurmountable obstacle. Consider the following:

Ankle A subject with a broken left ankle is sitting watching television. She is *TYP* free even though her ankle is broken. She rises from her chair, without putting any of her body weight on her left foot; nevertheless she feels a little *TYP*. Once she is upright, she tests her ankle by putting more and more weight on her foot, and feels a corresponding steady increase in the intensity of her *TYP* until the intensity of her experience drives her to lift her foot from the floor and sit back down. The *TYP* almost immediately eases, but it takes some ten minutes or more for her discomfort to disappear completely.

It is important to point out that this is not an exceptional experience. Normally *TYP* experienced by subjects with tissue damage comes and goes and it also ebbs and flows in intensity. According to TD perceptualism changes in the intensity of this

[14] I discuss this in greater detail in chapter 4, section *4.4.2*.
[15] See chapter 1, section *1.2.5*.

subject's *TYP* represent changes in the severity of tissue damage. Yet it is reasonable to assume there has been no corresponding objective change in the subject's physical condition; the severity of the fracture remains more or less constant throughout. Before she rises she has no *TYP* so the fracture is not being perceived (this is an example of perceptual failure – P5 above). Presumably, her slight *TYP* has the content that the damage is slight. Given, that she has a fracture this would be a non-veridical experience. But is *TYP* veridical when she puts her foot to the floor and *TYP* intensifies, when the *TYP* is at its peak just before she sits down, or at some point in between? No answer to this question will help TD perceptualists, because *Ankle* shows that the intensity of *TYP* and severity of tissue damage fluctuate independently of one another.

I can think of only two responses to this problem. Both take the assumption that *TYP* intensity represents the severity of tissue damage to be mistaken. The first is due to Christopher Hill. He proposes that *TYP* of a particular intensity has disjunctive content representing the severity of damage *and* the temporal characteristics of the damage; i.e. the length of time that the damage has existed (2005, p.95). He presents this solution as a simple disjunction, "either there is very recently incurred damage of level *N* or there is damage of degree *N** that has existed for interval *I*" (2005, p.95). To clarify this, damage of level *N* must have existed for a time so Hill is presenting damage of severity *N* for interval *I* or damage of severity *N** for interval *I′* as the disjunctive content of a *TYP* at intensity 3 (say). But why think that *TYP* at a given intensity has only two different contents? Once disjunctive content is invoked, Hill needs a principled reason to restrict the number of contents. Otherwise, a given

intensity of *TYP* could represent all possible combinations of damage severity and temporal intervals, just as another intensity of *TYP* could represent all possible combinations of damage severity and temporal intervals. There is no obvious solution to this problem.

Even if a token *TYP* is taken to have only two contents, Hill's disjunctive position has an odd consequence. On the stipulation that one content is accurate it follows that the other content is inaccurate, so the experience is both veridical and non-veridical. This problem is accentuated if pains are conceived to have more than two contents. Fortunately, I have no need to unravel the theoretical implications of these difficulties because Hill's disjunctive account is plainly *ad hoc*, and it does not solve the problem. Take *Ankle* as a plausible example of what might happen if you attempted to weight-bear on a fractured ankle. Hill would, I presume, explain the variations in *TYP* in terms of variations in disjunctive content. Which, if any, of these *TYP*s is veridical? On the *most* charitable view only one intensity of *TYP* could be veridical because there are no significant differences in either the severity of damage or the time that the damage has persisted during the course of *Ankle*.[16] Therefore, *at best* all but one of these different pains is non-veridical. So disjunctive content cannot solve the problem posed by P6.

The second response is to deny that the intensity of *TYP* represents the severity of damage. The idea here is that the intensity of *TYP* is like the brightness of a visual experience. The intensity of light that stimulates the photoreceptors in the eye is

[16] Unless there is overlapping content; i.e. two qualitatively distinct pains have the same content. I dismiss this interpretation because it opens Hill up to the charge that any token pain has content representing all possible intensity/temporal combinations of tissue damage.

causally related to the brightness of visual experiences. In this way, the objects we perceive look brighter, but we do not usually think of these variations in the intensity of our visual experiences as representing a property of the perceptual objects. This is reflected in natural sounding reports like, "I can see the words more clearly now because it's so much brighter than it was", which reflect a commonsense acceptance that visual experiences vary in brightness. Science supports this commonsense distinction between the stimulus and the perceptual object. We can see *objects* like words, hedges and swimming pools because they reflect light onto the retina of the eye. This light *stimulates* photoreceptors. Might the same be said of *TYP*?

For the sake of clarity, at the beginning of *1.2.5* I said I was taking tissue damage to be the stimulus. The evidence of pain science is that this false. The sensory nerves associated with *TYP* (called 'nociceptive neurons') express receptors (called 'nociceptors') which detect particular intensities of particular types of energy. They do not have the capacity to detect tissue damage.[17] So like visual experience there is a distinction between the perceptual object (tissue damage according to TD perceptualism) and the stimulus, which is energy. However, despite the apparent parallel this strategy will not work.

One problem is that tissue damage varies in severity. By this defence, this feature of tissue damage would not be part of the content of *TYP*. If skin damage is the perceptual object the severity of the damage might be perceived *visually* as the physical extent of the damage, but the severity of damage to deeper tissues like

[17] I present a detailed account and analysis of the 'nociceptive system' and its role in pain in chapters 3 and 4.

muscle, ligaments and so on would not be part of the content of *any* perceptual experience. Yet locutions like "I'm sure the sprain's worse because my ankle hurts a lot more than it did earlier" sound perfectly natural. Here the subject is taking the intensity of *TYP* to be, at least, an indicator of the severity of her tissue damage. We do not talk of visual experiences in the same way. The severity of a skin wound is perceived to be of a certain size because it is represented by phenomenal qualities that are distinct from the phenomenal qualities representing the surrounding undamaged skin. But we do not take the brightness of a visual experience to be an indicator of the severity of damage or the intensity of swimming pools or hedges. Indeed, we would deem it ridiculous to think that perceptual objects like swimming pools and hedges vary in intensity because our visual experiences vary in brightness. In short, we distinguish the brightness of our visual experiences from the perceptual object, but we link the intensity of *TYP* to the severity of damage. This asymmetry would need explaining.

Finally and critically, the introduction of peripheral neurology to the analysis has disastrous consequences for TD perceptualism. For visual experience, light is reflected off the perceptual object onto the retina so there is a direct causal chain leading from the perceptual object to the stimulation of photoreceptors. This is not the case for *TYP*. Nociceptors are defined as high-threshold receptors that detect *energy* intense enough to damage or threaten damage to the tissues.[18] The important thing to note here is that the energy has *two* causal effects, it stimulates the sensory receptors associated with pain and it *damages* tissue. There is no direct causal chain leading

[18] This definition is derived from the IASP's definitions of a 'nociceptor' and a 'noxious stimulus'. See chapter 4, section *4.1*.

from tissue damage to the stimulation of a nociceptor; i.e. tissue damage is not detected by any sensory receptors.[19] Nor are tissue damage and the stimulation of sensory receptors are linked by a common antecedent. The damaging of tissue is an event; tissue damage is the state that is caused by the event. Once the event is over, the receptors are not being stimulated but the damage remains. In this light, it is hardly surprising that *TYP* is commonly experienced in the absence of tissue damage (P4), that *TYP* is most often absent in the presence of tissue damage (P5) and there is not a one to one relationship between the severity of damage and the intensity of *TYP* (P6). TD perceptualism is unsustainable, but these objections point the way to a version of perceptualism that can handle many of the problem cases I have presented as evidence for P4-P6.

2.1.3 Perceptualism about pain – a revision

There are no good reasons to adopt TD perceptualism. Perhaps it might be pointed out that we tend to believe we have tissue damage when we experience *TYP* so it is commonsense to think of tissue damage as the perceptual object. But this observation has to be balanced against our ready acceptance that damaged tissue is not constantly painful, that pressing a wound is usually painful and that *TYP* inhibits any tendency to stretch a finger too far into extension. Indeed our commonsense expectations suggest a tacit recognition that *TYP* is causally linked to intensities of energy. So

[19] This is a little too quick. My use of 'direct' reflects the sensitising effect of the substances released by damaged tissue. As a consequence sensory neurons have lower thresholds in the presence of some tissue damage. This means that they transmit impulses at lower intensities of energy in the presence of tissue damage. But the sensitising of receptors is distinct from stimulation that directly causes a neuron to fire off impulses. This is discussed in much greater detail in chapter 4.

commonsense does not run counter to the view that *TYP* is a representation or an awareness (in the veridical case) of energy intense enough to damage or threaten damage to the tissues (i.e. *TYP* represents 'noxious' intensities of energy). For the sake of brevity, I call this version of perceptualism '*NE perceptualism*'.

NE perceptualism has the advantage of consistency with peripheral neuroscience. The IASP define the class of sensory receptor that is associated with *TYP* in terms of its ability to detect harmful or potentially harmful ('*noxious*') intensities of particular types of energy (IASP, 2014). These receptors are called '*nociceptors*' after the Latin word '*nocere*' for harm. Although it is not always clear from the literature there is a strong consensus that nociceptors are *adapted* to detect thermal or mechanical energy (see for example, Fields and Levine 1984, Mantyh et al 2002).[20] So NE perceptualism is more precisely characterised as:

NE Perceptualism A token of *TYP* is a representation or an awareness (in the veridical case) of a particular type (thermal or mechanical energy), bodily location and intensity of energy that is damaging or threatening damage to tissue.

Unlike TD perceptualism, NE perceptualism can provide satisfactory explanations for many of the problem cases I presented under P4-P6. Take everyday examples of *TYP*

[20] The ambiguity is associated with the distinction between the types of energy that can stimulate nociceptors and the types of energy that nociceptors have been adapted to detect. On the one hand "nociceptors can be activated by various forms of energy including mechanical, electromagnetic, electrical, thermal and chemical stimuli" (Coutaux et al, 2005). On the other hand it is extremely doubtful that nociceptors have been specifically adapted to detect electromagnetic and electrical energy. It is also questionable whether nociceptors have been adapted to detect chemical energy but these details are of little importance to my thesis. Here I take thermal and mechanical energy to be the relevant types of energy detected by nociceptors.

in the apparent absence of tissue damage (P4) like sitting for longer periods, stretching a finger into extension or pulling hairs on the back of a hand. NE perceptualism predicts that in each case, mechanical energy has reached or passed a threshold marking the division between innocuous and threatening intensities of energy (the '*noxious threshold*').[21] It also predicts that *TYP* will resolve once the intensity of energy is below the noxious threshold. The imposition of threatening intensities of energy also explains why we are most often *TYP*-free when we have tissue damage (it explains everyday cases of tissue damage in the absence of *TYP* – P5). In *Ankle*, the subject with the fracture is *TYP*-free when she is sitting because the forces acting at the site of her partially healed fracture are not being represented as noxious. But when she stands and puts her foot to the floor the intensity of the energy in her ankle increases and she feels *TYP* when the mechanical energy is represented as noxious. With respect to the case of the man warming his feet by a fire, NE perceptualism predicts the following:

*Fire** As the temperature in the man's right foot reaches the noxious threshold of about 42°C he would begin to feel slight *TYP*. This *TYP* might be ignored or pass unnoticed, in which case it would increase in intensity as the temperature in his tissues rises towards the damage threshold of 47°C. Consequently, he

[21] It is difficult to specify the noxious threshold precisely. The evidence is that the *damage threshold* to thermal energy is 47°C, for example. This is the threshold at which bodily tissue begins to damage. Clearly then thermal energy at 46°C cannot damage tissue. However, energy at 46°C might well intensify to damaging levels so it would be advantageous to experience pain when the temperatures in the tissues are at 46°C. The same might be said of 45°C, but what of 44°C or 43°C? The apparent arbitrariness of the concept of a noxious threshold can be avoided by consideration of pain thresholds. In short, we begin to experience pain when temperatures are around 42°C so an NE perceptualist might say this is the noxious threshold because we begin to represent thermal energy at this intensity as a threat. There are further degrees of complexity here. For example, once tissue is damaged it is vulnerable to lower intensities of energy. I discuss these matters in chapter 4 and chapter 6 section *6.2.1*.

> would be likely to remove his foot from the hearth. If for some reason he did not behave appropriately and burned his foot slightly as a consequence, his *TYP* would diminish once he removed his feet from the hearth because the intensity of the thermal energy in his foot would fall below the noxious threshold.

Unlike the prediction generated by TD perceptualism, this is a reasonably accurate account of what is likely to happen in actual circumstances.

Although NE perceptualism is explanatorily superior to TD perceptualism, the problem cases I presented in P4 and P5 remain difficult. I cited chronic problems, like the low back or neck *TYP*s, tension headaches and trigeminal neuralgia as examples of *TYP* in the absence of tissue damage,[22] but they are also examples of *TYP* in the absence of noxious intensities of energy as are complex regional pain syndrome (CRPS) and phantom limb pain (PLP).[23] If, as Murat Aydede suggests, cases like these were commonplace they would be a significant problem for NE perceptualism. However, his claim that "40% of all Americans suffer from chronic pain at least once and usually late in their lives" (2009, p.537, fn.7) is suggestive of rarity rather than the commonplace.[24] The implications of this statistic are that 60% of Americans do not suffer chronic *TYP* and of those that do only a small proportion of the *TYP*s they experience during their lives are of the problematic type. Given their rarity it seems to me that NE perceptualists can reasonably account for these chronic cases as non-veridical experiences. Of course, it might be objected that these errors are

[22] In chapter1, section *1.2.5.*
[23] CRPS is an ill-defined condition characterised by prolonged (often in a limb).
[24] See also fn.43, chapter 1.

commonplace when compared with visual hallucinations, but then the nature of the perceptual object of *TYP* must be considered. Noxious intensities of energy have the capacity to damage nociceptors and nociceptive neurons so defective detection and transmission are to be expected. Moreover, scientific explanation of chronic cases is consistent with this defence. According to pain science, many of these chronic syndromes are due to damage and maladaptive plasticity.[25] So NE perceptualism can cope with these cases of *TYP* in the absence of noxious intensities of energy, but neurological damage and maladaptive plasticity cannot explain the *occurrence* of injury in the absence of *TYP* in contexts like sport and war.[26]

Cases like these do not seem amenable to treatment as perceptual failures because the absence of *TYP* in circumstances like these is most persuasively explained as a normal (adaptive) process. Given the (sometimes extremely) unpleasant nature of *TYP* the suppression of the conscious perception of very intense noxious energy[27] in circumstances where *TYP*'s capacity to distract its subject could compromise survival is clearly adaptive. Scientists have identified several mechanisms that are involved in the inhibition of nociceptive input. The details of these mechanisms are not of relevance here, the point is that the absence of *TYP* is *normal* in such cases. So we have persuasive evolutionary (functional) and neurological explanations for the

[25] Maladaptive plasticity requires a little explanation. Like most anatomy and physiology, neuroanatomy and neurophysiology is adaptable. Normally, this adaptation is beneficial but occasionally it has the harmful effect of causing pain in the absence of noxious stimuli; i.e. the experience of pain at inappropriate times is the harm, hence 'maladaptive'. (For more on plasticity see Flor et al, 2006; and Woolf and Salter, 2000.)

[26] I have highlighted 'occurrence' to make it clear I am referring to an event, the damaging of a tissue by an intensity of energy exceeding the damage threshold for that tissue, rather than a tissue state (the damaged tissue). By NE perceptualism, the noxious energy involved in the former event should be perceived as pain, but the tissue state is never perceived as pain.

[27] I will occasionally abbreviate 'noxious intensities of energy' as 'noxious energy'. In this context, the term clarifies 'very high intensities of noxious intensities of energy'.

absence of *TYP*, but these explanations are not easily translated into the terms of perceptualism. The difficulty lies in explaining why *a perception* of noxious intensities of energy has the capacity to distract its subject to such an extent that she may fail to attend to matters of greater import. I will discuss this matter in the following section so for the moment I will simply deflect the issue by pointing out that cases like these are relatively rare. NE perceptualism faces a greater challenge rationalising the accuracy of *TYP* with the evidence of a variable relationship between the intensities of *TYP* and the stimulus (P6).

The science is that noxious intensities of energy stimulate specifically adapted sensory receptors called 'nociceptors'. So the evidence that there is not a one to one relationship between the intensities of *TYP* and the stimulus[28] is evidence of a weak correlation between the intensity of *TYP* and the noxious intensity of energy. Consider the following:

Sprain A subject with a sprained left ankle has been sitting watching television for a couple of hours. She is *TYP*-free even though her ligaments are sprained. She rises from her chair. In so doing, she puts a very small amount of body weight on her left foot and experiences quite intense *TYP* as a consequence. Nevertheless, she carries on and begins to limp around the room. The continued movement is accompanied by a steady decrease in the intensity of her *TYP* and so she slowly increases the intensity of the weight-bearing force on her ankle. After six minutes or so her *TYP* has eased to mild discomfort

[28] This is P6, chapter 1 section *1.2.5.*

> despite the fact that she is taking most of her body weight on her left foot as she walks. Soon she sits down again, but almost immediately the telephone rings and she stands again. This time her ankle *TYP* is very slight.[29]

According to NE perceptualism the absence of *TYP* when the subject is sitting means she is not perceiving a noxious intensity of energy in her ankle. This corresponds with the likely fact that the intensities of mechanical energy (mostly gravity acting on the tissues and internal pressure) at and around her sprain are below the noxious threshold. When she rises and first puts weight on her ankle the intense *TYP* she experiences is a perception that mechanical energy has exceeded the noxious threshold to a significant degree.[30] This too can be explained as a likely consequence of imposing some of her body weight on her sprained ankle. But what of the *TYP* that diminishes in intensity little by little as she limps around. Here the perception is that the intensity of energy is less noxious than it was on first standing and it is getting less and less noxious. This could be explained if she was reducing the amount of body weight she was imposing on her ankle, but the mechanical demands are increasing. Cases like these are difficult for NE perceptualism and they cannot be dismissed as rare anomalies because they are commonplace when tissue is damaged.[31] For example, individuals with low back *TYP* often experience severe *TYP* when rising from sitting. This is frequently sufficient to make standing upright and moving around

[29] This appears in chapter 1, section *1.2.5*. It is an important objection to NE perceptualism so I have repeated it here

[30] It is worth reminding the reader that this does not imply she is perceiving that the energy has exceeded the *damage* threshold.

[31] This claim is backed by the twenty years I spent working as a physiotherapist.

very difficult but after moving for a few minutes, the *TYP* often diminishes to the point that standing upright and walking are *TYP*-free.[32]

These examples of the variable relationship between the intensities of *TYP* and the intensity of the energy are particularly challenging because the variability could plausibly have gone the other way; continued (or repetitive) imposition of a *consistent* intensity of mechanical energy might be correlated with increasing intensities of *TYP*.[33] For example, the intensity of *TYP* might well have increased as the subject hobbled around on her sprained ankle. And the same might be said of *Ankle*. An NE perceptualist might be tempted to point to the IASP's definition of a noxious stimulus as a "stimulus that is damaging or threatens damage to *normal* tissues" (IASP, 2014; my emphasis), claiming that the concept of a noxious stimulus only applies to undamaged tissue. As my illustrations involve damaged tissue they are not cases involving noxious stimuli. But construed like this NE perceptualism would apply to only a proportion of *TYP*s; i.e. those that are perceptions of stimuli that are damaging or threatening damage to *normal* tissue. As such NE perceptualism would entail a disjunctivism about pain or that *TYP* in the presence of tissue damage is not pain. Neither position is appealing.

I will consider *Sprain* in the final section of this chapter, but there is little more I want to add here. When it comes to explaining P4-P6, NE perceptualism is the best version

[32] *Sprain* is an important illustration precisely because it is a commonplace example of the variable relationship between the intensity of pain and the stimulus and because it is difficult to explain. I explain circumstances like these in chapter 6, section *6.3.3*.

[33] Cases of increasing pain with no increase in the intensity of mechanical energy are amenable to explanation in terms consistent with NE perceptualism. When a given intensity of mechanical energy is repeated over time, the threat can increase; hence a given intensity of mechanical energy at a given bodily location can be innocuous at one time and noxious at another. See chapter 4, section *4.4.3* for more on this.

of perceptualism, even though it is not entirely satisfactory. Given the difficulties associated with P6 in particular, some perceptualists might be inclined to adopt a 'placeholder strategy'. By this strategy, *TYP* is the perception of something but as it is not clear what that something is, the perceptual object is not specified. This is a way of avoiding the problem posed by the correlation between *TYP* and either tissue damage or noxious intensities of energy. Presumably, someone adopting this approach would believe that *something relevant* is more closely correlated with pain than noxious intensities of energy. But what might that be? Physiological activity in some neurological structures almost certainly correlates closely with *TYP*, but the perception of this activity would not seem to provide an explanation for the injury-preventing function of *TYP* (P3). Current pain science provides little or no justification for the belief that anything other than sub-personal neurological activity correlates more closely with *TYP* than noxious intensities of energy so perceptualists would do well to adopt NE perceptualism. It is consistent with the nociceptive concepts of pain science and it would be difficult to explain the injury-preventing function of *TYP* if we were not able to perceive noxious intensities of energy. Consequently, in the coming section the perceptualists I consider will be treated as if they were NE perceptualists. So henceforth I use 'perceptualism' for the thesis that *a veridical TYP is a perception of the type, intensity, and location of energy which is damaging or threatening damage to the bodily tissues*. In the next sub-section, I will consider whether NE perceptualism can explain the compositional structure of *TYP* in perceptual terms that are consistent with an injury-preventing function.

2.1.4 Perceptualism – The composite structure and function of pain

In chapter 1 section *1.2.1*, I presented the *prima facie* view that *TYP* is a unitary experience constituted by characteristically unpleasant phenomenology as a problem for theorists of all stripes. On the one hand, the varied bodily locations, intensities and qualities of *TYP* seem amenable to explanation in perceptual but not motivational terms. On the other hand, the unpleasantness of *TYP* seems affective; we are averse to *TYP* and are motivated to avoid or ameliorate it as a consequence.[34] So *TYP* appears to be a unitary experience with *intrinsic* perceptual and motivational features unlike the phenomenal qualities of vision and audition, for example. In this light, all theorists have reason to welcome the evidence that *TYP* is a composite experience constituted by an affectively neutral phenomenal quality (a token of *Q*) and a further affective mental state (*U*) that makes it feel *as if Q* is intrinsically unpleasant.[35]

Some of this evidence comes from case studies in which subjects identify experiences as pain but also report that these experiences are not unpleasant. I have argued that on balance we have good reasons to take these reports at face value, that is to take it that these subjects are identifying experiences in a manner consistent with a recognitional concept they held prior to brain damage (or taking narcotic drugs). To recap, these subjects are identifying affectively neutral experiences as pain under *REC*; *an ability to categorise an occurrent experience as pain by reference to her memories of experiences she has learned to categorise together as pain*.[36] But recognition does not

[34] I am ignoring problem cases here, e.g. masochistic contexts and circumstances in which a subject believes that pain is the first sign of recovery from paralysis (say).
[35] This evidence is presented in the first part of section *1.2.2*, chapter 1.
[36] I introduce and explain *REC* in chapter 1 section *1.1*.

imply that these affectively neutral experiences *are* pains unless it is either assumed that pains are the experiences that could be identified as pain under REC (this is *T* in section *1.2.3*, chapter 1) or that something objective unites the experiences that are exhausted by *Q* and identified as pain under *REC* with the composite experiences constituted by *Q* and *U* that are identified as pain under *REC* (*TYP*). Perceptualism seems an appealing way of settling this matter. According to perceptualism, *TYP* has *unique* perceptual content. *TYP* is *the* type of experience that provides conscious information about noxious intensities of thermal, mechanical and chemical energy. In accordance with this unique content, *TYP* or a component of *TYP* has proprietary phenomenology.[37] Our ability to identify experiences as pain (*REC*) is explained by this proprietary phenomenology; we are able to identify the appropriate experiences as pain because they have the unique feel of pains. If an experience with some other content were to be identified as pain, it would be a mistake because the subject would not be deploying *REC*.

Now given the arguments and analysis on the constitution of *TYP* I conducted in sections *1.2.2* and *1.2.3* in chapter 1 it is reasonable for a perceptualist to claim the following:

Premise 1 The typical experiences we identify as pain are composite experiences constituted by a token of *Q* (a token of varied affectively neutral

[37] 'Proprietary phenomenology' means that pain owns its phenomenal character; it does not share this character with any other types of experiences. Pain would have proprietary phenomenology either if one of its components is a phenomenal quality that is uniquely for pain or if pain is uniquely constituted by components that individually constitute other (non-pain) experiences.

phenomenal qualities) and *U* (an affective mental state that makes it feel as if *Q* is intrinsically unpleasant).

Premise 2 Those with asymbolia identify unitary experiences constituted by *Q* as pain.

Premise 3 Normal subjects and those with asymbolia deploy the same recognitional concept (*REC*) when they identify experiences as pain.

And from the argument I have presented in this section:

Premise 4 The ability to identify experiences as pain under *REC* is explained by the proprietary phenomenology of pain.

Premise 5 The proprietary phenomenology of pain is explained by unique content about the type, location and intensity of noxious thermal and mechanical energy.

These premises yield the following conclusion:

Conclusion *Q* bears the perceptual content that individuates pain.

This supports the claim made by Bain, Grahek, Hall and Klein that an experience of *Q* is sufficient for pain. In response to this argument, it might be objected that premises 4 and 5 beg the question. If perceptualism is the thesis that *TYP* uniquely has content about noxious intensities of energy, then it requires proprietary phenomenology. To see this consider that if this were not the case a token of *Q* bearing content about

something other than noxious energy would be indistinguishable from a token of *Q* with such content. This would be a very good reason to doubt the testimony of people with asymbolia. Proprietary phenomenology plugs into premises 1, 2 and 3 to give the conclusion. I have provided reasons to accept that *TYP* has unique content (see the preceding section, *2.1.2*). Whether or not these reasons are good enough is central to the case for both perceptualism and Bain's, Grahek's, Hall's and Klein's view that an experience constituted by *Q* is sufficient for pain. However, this is not an issue at this point because my intention is to develop the most plausible version of perceptualism.

So given the conclusion that *Q* bears perceptual content which individuates pain, it is open to both perceptualists and mixed theorists to claim that *Q* is perceptual in nature and that an experience of *Q* is necessary and sufficient for pain. But by contrast with mixed theorists who face the apparently straightforward task of explaining *U* in motivational terms, perceptualists must also account for *U* in perceptual terms. Bain (2013), Cutter and Tye (2011),[38] and Grahek (2007) are all perceptualists. Although there are subtle differences between their positions, these subtleties do not matter here because I will argue that the problem is generic; it is the problem of providing a consistent pair of contents for *Q* and *U*.

2.1.5 Perceptualism – The content of U

I want to begin by making it clear it would be implausible for perceptualists to claim that the content of *U* has little or nothing to do with the content of *Q*. Subjects with

[38] Tye (2005a, b) also adopts this position.

brain damage (subjects with asymbolia and lobotomised subjects) and those on narcotic drugs are the only examples of the dissociation of *Q* and *U* cited by theorists. So it is reasonable to conclude that experiences of *Q* in the absence of *U* are abnormal.[39] Therefore, for all that has been said here, in normal experience *Q* is always experienced as part of a composite that has *U* as a constituent. The gestalt-like structure of *TYP* can only be explained by perceptualists in terms of perceptual contents (of *Q* and *U*) that are related in some way. Any other explanation would be implausible.

The perceptualism of Bain, Grahek, and Cutter and Tye differs, but each is a version of TD perceptualism. So I begin this sub-section by rationalising their accounts of *U* and the relationship between *Q* and *U* with perceptualism (with NE perceptualism). Bain and Grahek specifically consider the evidence that *TYP* has dissociable components. According to Bain's 'evaluativism'[40] these typical experiences are constituted by *Q* which represents tissue damage and a layer of evaluative content (*U*) which represents the damage as bad for its subject (2013, p.S82; 2014 p.306). Grahek's position is ambiguous. He seems to take the position that the sensory component (*Q*) represents tissue damage, but does not represent the 'force' or 'meaning' of the damage *to its subject*. Hence "the feeling of pain [*Q*] cannot, when taken alone, be understood as the perception or representation of bodily or tissue damage" (2007, p.80). It is difficult to know what to make of this, but the idea of representational *force* is not inconsistent with Bain's evaluativism and so I will

[39] One of the consequences of my overall position is that experiences constituted by tokens of *Q* in the absence of *U* are commonplace. (See chapter 5.)
[40] Norton Nelkin also calls his subtly different position 'evaluativism'. Nelkin accounts for *U* in terms of desire (1994). I interpret Bain's position as being closer to Bennett Helm's evaluation of the import (for the subject) of an injury (2002)

interpret Grahek in this way.[41] By contrast, Cutter and Tye make a conceptual distinction between *content* representing tissue damage and *content* representing that the damage is bad for its subject, but their position is ambiguous with respect to the evidence that *Q* and *U* are dissociable. Although, it is not entirely clear whether they would endorse the view that *Q* represents tissue damage and *U* represents that the damage is bad for its subject, this is the cleanest way of interpreting their position. So I take it they would broadly support the thumbnail characterisation I have derived from Bain's and Grahek's positions. For convenience, I will refer to the combination of TD perceptualism and evaluative content as '*TDE*'.

What does it mean to say that *U* represents that tissue damage *is bad* for its subject? Cutter and Tye equate 'being bad for you' with 'being apt to harm you' (2011, p.99) and Bain is open to specifying the content of *U* in the same terms (2013, p.S82). However, this phrase hardly clarifies matters. On an ordinary understanding, 'apt' means 'suitable' or a 'tendency'. Maybe it makes sense to describe tissue damage as suitable to cause harm or that tissue damage has a tendency to cause harm, but it is not obvious how this suitability or tendency might be represented by *U*. Part of the problem is that damage is normally equated with harm so necessarily tissue damage is a harm. But suitability and tendency sound contingent; tissue damage may be a harm. In my view, the equation of tissue damage with harm is a little misleading. Certainly all tissue damage is a harm, but the *extent* and *severity* of damage are distinct. Consider a wound of a certain extent *close* to the location of the great saphenous vein

[41] Bain would disagree. In his 2014, he explicitly distances himself from Grahek's position, but his interpretation of Grahek is questionable. On his reading Grahek takes *Q* as a representation of a harmful stimulus and *U* as a representation of the stimulus as damaging or threatening damage (2014, p.310). This sounds tautologous so on Bain's reading, the content of Grahek's composite experience is overdetermined. I doubt this was Grahek's intention.

and a wound of the same extent *at* the location of the great saphenous vein.[42] The latter is more severe than the former because it is a greater threat to the subject. The relationship between the extent and severity of tissue damage is complex. It can involve what might be termed primary threats like that in the example above, fluid loss and the length of healing time, as well as well as secondary threats such as a compromised ability to weight bear due to the location and extent of damage (e.g. a patella tendon strain). Hence, tissue damage of a certain extent at a particular location has a *potential* to harm in various ways, where harm implies compromised function.[43] So TDE is the position that *Q* represents the type, location and extent of tissue damage, and *U* represents the potential harm to the subject of the damage represented by *Q*.[44]

The immediate problem with TDE is that TD perceptualism is unsustainable; *Q* cannot represent tissue damage for the reasons set out in sub-section *2.1.1*, above. The trouble is that content about noxious energy (the content of *Q* according to NE perceptualism) does not sit easily with evaluative content about the potential to harm. The idea that *U* represents the potential harm to the subject of the damage caused by the noxious intensities of energy represented by *Q* will not work because tissue damage would have to be represented by *U*. Consider that TDE is the thesis that *U* is an evaluation that the content of *Q* is apt to harm. The content of *Q* does not refer to

[42] Cutter and Tye use this example, though they present it as an obstacle to their position. The problem being that both cases are likely to feel more or less equally unpleasant, and so they are represented as more or less equally bad (2011, p.102). I will not address this objection here.
[43] Bain (2012, p.S82) and Bennett Helm (2002, p.23) both put it more or less this way.
[44] The scientifically acceptable concept of salience could have been used here; *U* represents the salience (the relevance for the subject) of the damage represented by *Q*. The trouble with salience is that it does equal duty for both pleasant and unpleasant experiences so it does not capture the unpleasantness (the bad experience) of *U*. In my view, harm is a more appropriate term and any misunderstanding can be resolved by equating harm with compromised function.

damage and yet by this idea, the content of *U* is about *the damage* caused by the energy represented by *Q*. This is a considerable problem. The evidence from P4-P6 counts against any *conscious* representation of tissue damage, and the evidence that no sensory receptors are specifically adapted to detect tissue damage suggests it is unlikely that tissue damage is represented at the sub-personal level. Now it might be responded that the latter does not preclude the possibility that we have a higher order perceptual system that takes inputs from multiple sources to produce the sub-personal perception that tissue is damaged to some degree. But even if this were a viable claim, the claim that *U* has content representing a sub-personal evaluation that tissue damage is apt to harm is implausible. This strategy would divorce *U* from *Q*, because the former does not refer to the content of the latter. Indeed, we would expect to experience *U* constantly when we have tissue damage. So *U* would normally be experienced in the absence of *Q*. That this does not happen is a very good reason to reject this understanding of *U*.

Alternatively, it might be thought that tissue damage is implied by the content that energy is *noxious*. Now it can be conceded that having the capacity to threaten or cause tissue damage to some degree *is* the property of being noxious, but the representation of this capacity is a representation that the intensity of a particular type of energy is at or exceeds the noxious threshold to some degree. This does not equate to a representation of damage caused. Remember, NE perceptualism is the position that *Q* represents *properties of energy*, its type, bodily location and that its intensity exceeds the noxious threshold to some degree.[45] Therefore, if *U* has content about

[45] The noxious threshold is the threshold at which an intensity of a given type of energy passes from being innocuous to being threatening. (See this chapter fn.21.)

potential harm and the content of *Q* enters into this content, then (in the veridical case) *U* is a perception of the potential harm to the subject of the noxious energy represented by *Q*. Where harm is the harm *to the subject* (i.e. a given intensity of energy that exceeds the noxious threshold to a given degree is more harmful for the subject at one location than another[46]) rather than the extent by which the energy exceeds the noxious threshold (this is represented by *Q*).[47] This important distinction avoids the problem posed by identifying harm with noxiousness. If they were identified with one another so that *Q* represents noxious energy and *U* represents harm, then the noxiousness of the energy is over-determined in *TYP*. Alternatively, if *U* were taken to represent noxiousness and *Q* to represent energy rather than noxious energy, tokens of *Q* would look to be indistinguishable from other token experiences which represent (innocuous) energy.

I have now arrived at a preliminary understanding of the components of *TYP* according to NE perceptualism

PQ - *Q* is a representation or an awareness (in the veridical case) of the type, bodily location of noxious intensities of thermal and mechanical energy.[48]

[46] This understanding faces an empirical problem; see below, this section.
[47] It is worth remarking that equating harm with being bad as Bain (2013) Cutter and Tye (2011) do is helpful because it conveys something of the feel that *U* contributes to *TYP*.
[48] In accord with the view of Bain, Grahek, Hall and Klein, I take it that *PQ* amounts to the position that tokens of *Q* are specifically for *TYP*, though it is of course possible that token phenomenal qualities with content about noxious intensities of energy are constituents of other experiences.

PU - *U* is a representation or an awareness (in the veridical case) of the harmful nature of the energy represented by *PQ*.

PQ is strongly supported by the 'nociceptive' concepts of pain science. According to these concepts nociceptors are sensory receptors that are specifically adapted to detect noxious intensities of thermal and mechanical energy. Usually activity in nociceptors and nociceptive neurons is causally related to experiences of *TYP*.

Although this understanding of *U* is initially appealing the nature of the harm expressed in *PU* needs a little development. If it is just taken to be related to location, like the great saphenous vein example above, then *PU* is seriously threatened by the variable relationship between the intensities of *Q* and *U* (P6). Clearly, damage of a given extent to both a right gluteus maximus muscle and an eye is much more harmful for the subject in the latter case. This gives a generalisation, energy at an intensity that is capable of damaging tissue to a given extent is more harmful in one location than another; i.e. *PQ* and *PU* vary independently of one another from location to location. But there would be an isomorphic relationship between the representations of the intensities of noxious energy and harm *at a given bodily location*. This is problematic because the evidence is that there is no isomorphic relationship between the intensities of *Q* and *U* at a bodily location (Rainville, et al, 1999). How then to explain this independence in a way that is sympathetic to perceptualism?

The independence of the intensities of *Q* and *U* seems amenable to explanation in evaluative terms, where the potential for harm of the noxious energy represented by

PQ is evaluated in relation to other potential harms to, and/or interests of, the subject. It is certainly the case that in some contexts the attention-grabbing unpleasantness of *TYP* could lead to greater harm because the subject might be distracted from a much greater threat. So more pressing matters trump the potential for harm that would be represented by *PU* in other circumstances.[49] Under *PU*, the low intensity or absence of *U* in such circumstances should be treated as a misrepresentation even though these fluctuations are, on this analysis, the product of a normal evaluative process. This thought leads naturally to the idea of embracing this normal process into the content of *PU*.

PU′ - *U* is a representation or an awareness (in the veridical case) of the harmful nature of the energy represented by *PQ* relative to other considerations.

According to *PU′* the perceptual content of *U* is extremely complex. Indeed it would be difficult to find an adequate way of expressing an example of this relative content. Nonetheless, expressed vaguely in terms of background conditions *PU′* can explain the independence of the intensities of *Q* and *U*; the independence of the intensities of *Q* and *U* is a function of background conditions. This position is scientifically respectable because of the evidence that peripheral and central processing of nociceptive input is modulated by cognitive and emotional influences like anxiety and

[49] 'Salience' is perhaps the best way of capturing the relativity of these competing concerns, but in my view salience does not adequately convey the unpleasant feel that *U* contributes to *TYP*.

memory (Tracey and Mantyh, 2007).[50] For now, it is enough to say that, for perceptualists, I believe that *PU′* is the most favourable way of accounting for *U*.

With a plausible perceptual account of the gestalt-like structure of *TYP* and scientific support for the crucial position that the phenomenology of *Q* represents noxious intensities of thermal and mechanical energy, perceptualism looks like a viable thesis. I now I turn to considering the relationship between perceptual content and the injury-preventing function of *TYP*.

2.1.6 Perceptualism – evaluative content and motivation

On the face of it perceptualism provides a strong explanation of asymbolia. Those with asymbolia are able to experience *Q*, but are unable to experience *Q* as if it were intrinsically unpleasant (they are unable to experience *U*). By perceptualism they are able to have experiences representing noxious intensities of energy at different bodily locations, but they cannot have experiences representing the harm posed by the energy represented by *Q*. As those with asymbolia suffer serious injuries when compared with normal subjects there is evidence that perceptual experiences (constituted by tokens of *Q*) in the absence of *U* do not adequately motivate injury-preventing behaviours.[51] So the evidence is that the content that the energy represented by *Q* is harmful fulfils a necessary link in the adequate motivation of injury-preventing

[50] The themes of processing, background conditions and modulation feature heavily in the next four chapters.
[51] My talk of '*adequately* motivating injury-preventing behaviours' is intended to convey the motivation of behaviours that provide a biological advantage.

behaviours.[52] A perceptualist needs to explain how this content links the content of *Q* with motivation, without opening herself to the charge that *U* is really a motivational mental state. At this point, it will be helpful if I introduce a conceptual framework that I will developed in chapters 5 and 6. This framework links inputs with nocifensive outputs like injury-preventing behaviours.[53]

Properly understood, the phenomenology of perceptual experience is a link in a causal chain leading to appropriate responses. On the *input* side, sensory information about the mind-independent world is causally related to phenomenal experiences which function as conscious perceptions of features of the mind-independent world. On the *output* side, perceptual experiences have the capacity to yield behavioural modifications that are advantageous for the perceiver. To put it another way, an ability to discriminate between the relevant and the irrelevant and between the relevant (the ability to perceive) and an ability to respond and behave in different ways are of no advantage to the perceiver unless discriminations can be linked with appropriate responses and behaviours. So visual phenomenology fulfils a perceptual function in systems that have the capacity to yield advantageous (functional) responses and behaviours. Pain scientists are concerned with all aspects of the functional system that is relevant to pain (henceforth, I will refer to this system as the '*nocifensive functional system*' or '*FS*' for short). The crucial issue is whether *U* is most plausibly conceived as an input or as an output of *FS*.

[52] The evidence provided by subjects with peripheral neuropathies is that experiences of *TYP* function to motivate injury-preventing behaviours (this is part of the evidence for P3 – see chapter 1, section *1.2.4*)

[53] The term 'nocifense' is widely used. The IASP refer to 'complex nocifensive *behaviour'* in their explanation of nociception (2014), but increased blood pressure, reflex withdrawal from a stimulus and pain are also nocifensive responses.

According to NE perceptualism, a token of *Q* is an affectively neutral experience that functions as a means of consciously representing (or as a conscious awareness of) the types and bodily locations of noxious intensities of energy. As *U* functions as a means of consciously representing (or as a conscious awareness of) the nature of the relevance of the content of *Q* given various (relevant) background conditions it appears that *U* follows *Q* in a serial manner; i.e. *U* represents an evaluation of the content of *Q*. Nevertheless, *U* is about the particular relevance of energy so it is a discriminatory experience. Conceptually, this locates the evaluative content of *U* on the *input* side of *FS*. But *U* is strikingly unlike the phenomenology of visual experience and the phenomenology of *Q*, which are also on the input sides of functional systems, because it adds immediate attention grabbing force to *Q*. Nothing in the expression of the content of *U* or its location in *FS* explains this immediate negative attention-grabbing force.

Both Bain (2013) and Helm (2002) attempt to address this issue by equating the evaluation that energy is noxious with motivation: to evaluate that energy is noxious is to be motivated. On Helm's terms, *U* would be a 'felt evaluation' of the import or badness of bodily injury (2002, p.24). His term 'felt evaluation' is used to mark a distinction with 'evaluative judgements', but his term could be misleading. The word 'evaluation' is often used to describe both a process and the conclusion of such a process. Contrast, "I am conducting an evaluation of its potential" with "My evaluation is that the project isn't worth pursuing". Clearly, Helm's position is that *U* represents the conclusion of a process of (presumably sub-personal) evaluation; that a

noxious intensity of energy is harmful, according to perceptualism. But these representations of evaluative conclusions are not affectively neutral, they make a token *Q* feel as if it were intrinsically unpleasant. This motivates us. So *U* straddles the divide between input and output. This is just the approach that Bain takes in his account of unpleasantness. He challenges the Humean assumption that informational states cannot motivate. We are motivated by the energy represented by *Q* precisely because it is evaluated and represented as bad (Bain 2013).

I do not find this position appealing. Bain needs this position or something like it to maintain his perceptualism, though perceptualism so construed looks at least a little like mixed perceptualism/motivationalism. In particular, an explanation is needed for why a sub-personal evaluation that energy is bad for its subject is represented at the conscious level. It is not obvious to me that this conscious evaluative content fulfils either any functional or explanatory role. The sub-personal evaluation clearly takes inputs and yields an output so it fulfils a linking role. Why think that it links input with an output that is both perceptual and motivational; i.e. a conscious output that functions as a representation of the sub-personal evaluation of harm *and* as a mental state that is (at least) necessary for the appropriate motivation of its subject. This representational function of *U* seems functionally superfluous. A mixed perceptual/motivational account is explanatorily and ontologically more economical because it does without a layer of redundant conscious perceptual content:[54] A sub-personal evaluation that noxious energy is bad triggers a conscious mental state (*U*)

[54] Aydede makes the same point, describing the former perceptual account as otiose by comparison with the latter account (2005b, p.132).

that functions as an immediate and forceful means motivating of injury-preventing behaviour.

This mixed position accommodates the background conditions (e.g. anxiety and memory, as well as contextual influences like sport or war) mentioned in *PU′* much more naturally than perceptualism. To see this consider that a particular type and intensity of energy at a particular bodily location is potentially *more* harmful for the subject in a life or death situation[55] so by *PU′* a mental state that functions as a representation of potential harm (*U*) should (in the veridical case) be more intense in such circumstances. This is contradicted by the examples cited under P5 (chapter 1, section *1.2.5*). By the alternative, there is no need to consider the accuracy conditions of *U*, because *U*, being motivational in nature rather than perceptual, does not have accuracy conditions. On this account, the sub-personal evaluative process integrates inputs from the nociceptive system with other perceptual and cognitive inputs (the background conditions) and 'decides' whether and to what degree it is appropriate to evoke an immediate and forceful motivational mental state (*U*).[56]

It is a considerable explanatory challenge to equate a conscious experience representing a discrimination with motivation. I do not think this challenge can be met. If it is accepted that *U* is evoked in response to the evaluation that noxious energy is bad, why think that this content is represented at the level of consciousness?

[55] Because the resulting damage is likely to compromise function to such a degree that the circumstances lead to the severe injury or death of the subject.
[56] Of course, the decision to evoke *U* can be assessed in terms of accuracy conditions, it is or is not appropriate to evoke *U* to some degree given the objective circumstances, but this content is the conclusion of the sub-personal evaluative process it is not a constituent of *U*, which functions purely as a means of motivating injury-preventing behaviour.

It is difficult to understand what function the conscious representation of such content could play in *FS*. The mixed perceptual/motivational alternative I set out seems preferable.

Perceptualists might respond by claiming that both the unpleasantness and motivational function of *TYP* are explained by perceptual content. Put graphically this response amounts to the claim that content representing the occurrence of bodily harm explains why *TYP* has a quality that makes it feel as though something harmful is happening to the body. And if something feels harmful we are very likely to be motivated by it.

All theorists can accept that in most (if not all) circumstances subject's are averse to or dislike the unpleasantness of *TYP*. They might also be inclined to agree that when a subject is experiencing *TYP* she tends to believe that something harmful is occurring to the body. But the claim that a subject holds a belief that something is harmful, which is grounded on a token *TYP*, does not imply that *TYP* has content representing harm. The issue here is not what we might believe *about TYP*, or whether harm (or better still 'badness') is a neat way of describing the unpleasantness of *TYP*. In the context of this thesis, these are trivial matters. The issue is functional; it is whether the ability to experience *Q* as if it were intrinsically unpleasant has evolved to function as a representation of an evaluation of harm. As this representation fulfils no obvious explanatory role in the motivation of injury-preventing behaviour there is a good reason to believe that *U* does not have this content. It remains to be argued that some other representational content fulfils such an explanatory role, but as I have treated

perceptualism as sympathetically as I can, my view is that the prospect for the specification of a content that would meet this challenge is slight.

My attempts to rationalise perceptualism with P1-P6 have ended up in a mixed perceptual/motivational position. This is hardly surprising as *prima facie TYP* is more amenable to a mixed rather than a unitary treatment. Indeed, I am unsure whether evaluativism really counts as a unitary position, given that *U* is motivating *because* it represents that a bodily disturbance is bad for its subject (2013, p.S82).[57] Perhaps then, accounts by representationalists like Bain and Tye actually concede some of the theoretical ground to mixed theorists. It is worth noting that other philosophers like David Armstrong and George Pitcher, who might otherwise be described as perceptualists are mixed theorists when it comes to *TYP*. The concession of ground towards the mixed position is also evident from the opposing direction as Richard Hall and Manolo Martinez, who Bain categorises as motivationalists, are mixed theorists by my lights.[58] This means that as far as I am aware, Colin Klein and myself, are the only philosophers who press motivational accounts of *Q* and *U*. In the following section, I will present and reject Klein's motivationalism before elaborating the mixed position in the final section of this chapter.

[57] Remember that bodily disturbances interpreted as bodily or tissue states are implausible candidates for the perceptual object of pain.

[58] Hall writes that pains are best viewed as having "compound intentional content, part descriptive and part imperative" (2008, p.534). Martinez gives an imperative account of the 'painfulness of pain' (2011). In my terms, I take this to mean that *U* has the (imperative) content "Don't have this bodily disturbance!" (2010, p.76). Note that perceptual content about a bodily disturbance is a constituent of his imperatival content.

2.2 MOTIVATIONALISM

The advantage of motivationalism is that it offers the prospect of a natural explanation for *U*, but it is not at all obvious how *Q* might be conceived in motivational terms. To recap, consider that subjects are motivated by composite experiences constituted by *Q* and *U* and that those with asymbolia are not motivated by experiences constituted by *Q* (in the absence of *U*) and they suffer a greater number of and more severe injuries than normal subjects as a consequence.[59] From this, it seems obvious that *Q* is affectively neutral while *U* fulfils an essential motivating role. This raises questions about the constitution of pain; is a token of *Q* necessary and sufficient for pain, are some pains constituted by *Q* and some by *Q* and *U*, or is *U* necessary for pain. Klein's position is interesting. He takes a token of *Q* to be necessary and sufficient for pain and he takes pain to be intrinsically *motivational* even though those with asymbolia are not motivated by experiences constituted solely by a token of *Q*. Their experiences are explained by a subject level deficit, a lost capacity to care.

Klein's argument is derived from his characterisation of motivation as "a two-place relation between a sensation and an agent" (forthcoming, p.13).[60] A token of *Q* (a pain on his account), which has an intrinsic motivational property (property *M*), is the first *relatum*.[61] The second *relatum* is a subject's capacity to care ('*CC*') about their body "in whatever way is relevant to pain" (forthcoming, p.6). So a normal subject is affected by her experience of *Q* because she has the capacity to care about her body in

[59] See chapter 1, section *1.2.4*.
[60] This reference to a 'sensation' is a little confusing.
[61] Property *M* is property *p* on Klein's account.

the relevant way. This capacity "requires hooking up sensation, cognition, affect, and behaviour in the right ways regardless of how one comes to know about a threat… asymbolics lack this integrative capacity, because their lesion has destroyed the neural substrate on which the capacity depends" (forthcoming, p.6). Hence he refers to his explanatory account of asymbolia as the 'lost capacity model' ('*LC*'). The implication being that the capacity to care is necessary for a subject to experience the motivational force of *Q* (to experience *M*). In short, Klein is saying that *Q* exhausts all the intrinsic properties of the experiences we typically recognise as pain.[62] Asymbolia is explained by an inability to experience all the intrinsic properties of *Q*.

To summarise Klein's motivationalism in my terms, *TYP* is constituted by *Q* and *U*, where *Q* has intrinsic motivational property *M* and *Q* is pain, and where *U* is a capacity to care about one's body. *Q* and *U* are necessary and sufficient for a subject to experience the unpleasantness that *appears* to be an intrinsic property of pain. My development of Klein's position in terms of motivationalism is questionable because, and he explicitly acknowledges this, his account lacks detail. This detail would help interpretation but importantly it is critical for the viability of his account.[63]

Klein suggests various candidates for *M*. Unsurprisingly he settles on imperatives, commands like "Stop putting weight on your ankle", as the most promising of these

[62] I have used this phrase rather than the retraction '*TYP*' here because I have characterised *TYP* in terms of two *mental states Q* and *U*. The latter is a mental state that makes it feel as if *Q* is intrinsically unpleasant. On Klein's account a *capacity* to care is necessary for a subject to experience *Q* as unpleasant. (See below).

[63] For the purpose of my thesis it does not matter whether my understanding of Klein's position accurately reflects his position. My aim in this section is to develop a strong version of motivationalism which is based on Klein's account.

(forthcoming, p.15).[64] At times he writes as though *Q* is exhausted by *M* and at others it seems as though *M* partly constitutes *Q*. Part of the problem here is that he *identifies* *M* with imperatives. If *M* is the imperative and the imperative exhausts *Q* (i.e. *M* and *Q* are identical), then it would seem that those with asymbolia would not experience *Q* because *CC* is necessary to experience *M*. But on Klein's (and everyone else's) account those with asymbolia experience *Q* so by Klein's lights *Q* and *M* are not identical. But then if *M* is identical with the imperative content of *Q*, *Q* is not exhausted by imperative content. In which case, he needs an account of the residue (the part of *Q* that is not *M*) that is consistent with his motivationalism. This is a significant explanatory problem.

In my view Klein's position is more favourable if imperative content is identified with *Q* rather than *M*. Metaphorically then, someone with asymbolia can hear the commands without registering their import or better, their *force*. This reading is consistent with the way he cashes out imperatives.

> The asymbolic recognises pains, because their ordinary imperative content has not changed. However, they have ceased to treat such bodily commands as binding, and so have ceased to be motivated by them (Klein, forthcoming; p.17).

[64] It is unsurprising because Klein is an imperativist about pain. Imperativism is a controversial thesis, but the details of imperativism, and its virtues and vices are not a concern here. I am assuming that a suitable candidate for *M* is available. The measure of his account is whether it can address P1-P6. Richard Hall and Manolo Martinez are imperativists about *U*, but they account for *Q* in perceptual terms so on my classification they are mixed theorists. (See Klein, 2007. See Bain, 2011 and Tumulty, 2009 for critical accounts of Klein's imperativism, and Klein, 2010 for a response to Tumulty.)

So the capacity to care is necessary for a subject to experience the mptivational force of an injury-preventing imperative. This force is identical to *M*. This way Klein can claim that *Q* is exhausted by its motivational function and resist the objection that asymbolia is a counterexample to motivationalism. Several problems remain though.

Klein's position seems vulnerable to the sort of Euthyphro problem that Bain sees as an obstacle to certain accounts of the unpleasantness of pain (2012, p.S80). We care about pain (or the content of pain – *Q* on Klein's account) because it is experienced as unpleasant; it is not that pain is unpleasant because we care about it. But I do not think this is too much of a problem. According to Klein a subject with the capacity to care experiences *Q* as unpleasant because she is able to experience the motivational force of the imperative content of *Q* (metaphorically, she can 'hear' the urgency of the command). The unpleasantness tends to trigger an aversive response. He can claim his bodily cares are at a different level to more generic cares; a subject cares about her *TYP*s because she is averse to their unpleasantness.

A greater threat comes from the objection that Klein's position is unmotivated; Klein needs *M* or something like it to sustain his motivationalism, but there is no good independent reason to accept his account. In response, Klein considers it a virtue that his *LC* model provides a strong explanation for the multiple ('non-pain') personal level deficits exhibited by those with asymbolia. This is a questionable advantage.

Indifference to the threat posed by the content of verbally and visually represented threats (Berthier, et al, 1988, p.43)[65] is an example of a 'non-pain' deficit exhibited by some with asymbolia. This is caused by a lost capacity to care about the body in the relevant way, according to Klein. The implication is that normal subjects (those with *CC*) would not be indifferent to these representations of threat. But for normal subjects the capacity to care about verbally and visually represented threats does not manifest itself subjectively as an experience with the immediate and forceful unpleasantness of *TYP*, even though *CC* is presumably fulfilling the same role for both *TYP* and verbal and visual threats. Why the subjective difference? Klein's likely response is that these threats lack *M*, which is unique to pain. But then if *Q* is motivational in virtue of *M*, both verbal threats and visual representations seem to require a different motivational property (call this '*B*'). So as *B* would be intrinsic to the *content* of a sub-population of auditory and visual experiences (i.e. those that represent a threat to the body) then we have a disjunctive explanation of phenomenal content. Note that the relevant content is perceptual.[66] Consequently, on the principle that *Q* is open to the same treatment, an opponent might claim that *M* is intrinsic to the perceptual content of *Q*. This has the advantage of being consistent with the scientific evidence that activity in the somatosensory cortex is linked with *Q*. In more detail, the science is that *Q* is subserved by sensory mechanisms and they link *Q* with activity in the somatosensory cortices, brain areas which are associated with sensory discrimination. By contrast, they associate motivational affect with activity in areas of

[65] See also Hemphill and Stengel, 1940.
[66] This seems an additional problem. Is a visual experience representing a bus bearing down on its subject representing a threat or is it representing the location (and speed) of a bus in relation to a perceiver? It is unclear where Klein would (or if Klein would) fit an imperative into an explanation of the perceiver running to avoid the bus.

the limbic system, like the anterior cingulate and insula cortices (the ACC and IC).[67] Klein's position would be stronger if he explained why activity in the somatosensory cortex is consistent with the motivational function he ascribes to *Q*.[68]

Another related problem is that Klein's invoking of *M* raises questions about how other affectively neutral (for the subject[69]) experiences motivate their subjects. The phenomenology of *Q* appears to be similar to the phenomenology of vision, audition, olfaction, etc. in important respects. It varies in quality, intensity and location, and crucially it *appears to be* affectively neutral just like visual, auditory and olfactory experiences. As *Q* requires *M* and *CC* for motivation it seems that these other experiences should be treated in the same way. The taste of chocolate motivates me to eat chocolate, but I also have the capacity to be satiated by eating chocolate. Satiation is consistent with a lack of motivation and if I continued to eat chocolate I might well experience a negative motivation, a drive to stop eating chocolate. If motivational properties are intrinsic features of phenomenal qualities does this mean that many phenomenal qualities have both prescriptive and proscriptive motivational properties and what mechanism would determine which (prescriptive or proscriptive) motivational property I am experiencing at any one time. And more problematically perhaps, how is indifference to be explained if motivation is a relation between a capacity of the relevant sort and an intrinsic property of phenomenal qualities? These difficulties seem to require some sort of masking mechanism to conceal one property

[67] Damage to the IC is claimed to be a causal factor in pain asymbolia (see chapter 1, section *1.2.1*).
[68] In chapter 5 I present a motivational account that is consistent with the sensory function attributed to the somatosensory cortex.
[69] 'For the subject' is intended to reflect Klein's view that the motivational property *M* is an intrinsic property of *Q* even though it is inaccessible to those with asymbolia. It is worth emphasising that it is not that *M* passes unnoticed or that subjects are not aware of *M*, it is that they cannot experience *M*. This makes it sound as though *Q* is intrinsically unpleasant even though *Q* is not experienced as unpleasant. To my mind this mix of the subjective and objective is unappealing.

or the other or both. The alternative is that different capacities are at play, but then these would need switching on and off. Intuitively this seems the better approach. Consideration of this approach reveals what I consider to be the main problem with Klein's motivationalism.

The capacity to like, dislike or be indifferent to chocolate is on a linear scale. Present me with chocolate and my preference is somewhere on that scale. No (*M*-like) properties need be ascribed to the affectively neutral phenomenal qualities associated with tasting chocolate or anything else. The motivational force is on the subject side of the motivational relation. *Q* and *CC* seem amenable to the same treatment. A token of *Q* is constituted by an affectively neutral phenomenal quality. A subject side disposition to be (negatively) motivated by tokens of *Q* is necessary for *Q* to be experienced as if it were intrinsically unpleasant. Furthermore, on this account *Q* is open to treatment in the perceptual terms that seem appropriate to flavours, visual experiences, and so on. This sort of treatment is consistent with the *prima facie* perceptual features of *Q* (varied qualities, intensities and locations), but it is not obviously consistent with imperativism. In this respect, the cause of imperativism specifically and motivationalism more generally is not helped by the subjective differences between what might be described as 'thermal and mechanical pains'. The subjective difference between the *TYP*s associated with picking up a very hot pan and stretching a finger into extension. "Let go!" or "Stop stretching now!" do not capture this distinction, but they are naturally accommodated by the *prima facie* perceptual account of *Q*.[70] The *TYP*s associated with these actions have perceptual components

[70] This is problem P1; see chapter 1, section *1.2.1*.

that represent the heat of the tissues absorbed from the pan and the mechanical stretch of the tissues.

Given these concerns, Klein needs to provide a reason for preferring his motivationalism to a perceptual treatment of *Q* that is independent of his need as an imperativist to ascribe *M* to *Q*. The independent reason he gives is that his lost capacity model has the power to explain the non-pain deficits of asymbolia. I have argued above that the details of this explanation has not been adequately worked out. But even if they were, his claim of advantage amounts to the claim that (specific?) insular damage affects the capacity to care *and* this explains *all* the deficits of asymbolia. The difficulty is that a unitary theory like this is not consistent with the multiple functions of the insula. The multiple deficits of asymbolia are consistent with multiple lost capacities. In conclusion, as it stands Klein's motivationalism cannot adequately address P1-P6, and in my view the prospect of an adequate explanation for these problems is bleak.

2.3 MIXED MOTIVATIONALISM/PERCEPTUALISM

In the penultimate paragraph of section *2.1.3* I outlined a mixed position that more or less drops out of the argument for perceptualism. According to this position, *Q* has perceptual content about the type, and bodily location of noxious intensities of energy, while *U* functions as an immediate and forceful means of motivating injury-preventing behaviour that is directed at the noxious energy represented by *Q*. These distinct mental states are linked by a sub-personal evaluative process that determines

whether and to what extent *U* is an appropriate response to the perceptual content, given certain background conditions. It is highly unlikely that the content of a conscious experience like *Q* fulfils a role in this evaluative process as *Q* would have to be evoked before influencing the evaluative process. As such this would be a serial process and serial processes are relatively slow. Furthermore, it is not clear how subjectively accessible content would feed into a sub-personal evaluative process. As *Q* and *U* are subserved by parallel pathways it seems probable that sub-personal content contributes to this evaluation.[71]

This position is explanatorily and ontologically more economical than NE perceptualism because it does without a layer of functionally redundant conscious perceptual content. It also accommodates the background conditions (e.g. anxiety and memory, as well as contextual influences like sport or war) mentioned in *PU′* much more naturally than perceptualism. To see this consider that a particular type and intensity of energy at a particular bodily location is potentially *more* harmful for the subject in a life or death situation so by *PU′* a mental state that functions as a representation of potential harm (*U*) should (in the veridical case) be more intense in such circumstances. By the alternative, there is no need to consider the accuracy conditions of *U*, because *U*, being motivational in nature rather than perceptual, does not have accuracy conditions. On this account, the sub-personal evaluative process integrates inputs from the nociceptive system with other perceptual and cognitive inputs (the background conditions) and 'decides' whether and to what degree it is

[71] There are also serial projections from the somatosensory cortices (the brain areas associated with *Q*) to the anterior cingulate and insular cortices (brain areas associated with *U*) (Fields, 1999). For more detail on these pathways see chapter 3, section *3.2*.

appropriate to evoke an immediate and forceful mental state that motivates injury-preventing behaviour.

The mixed position provides a natural sounding explanation for the individual components of *TYP*; *Q* is able to fulfil a perceptual function in *TYP* because it has the intrinsic features of a perceptual mental state and *U* fulfils a motivational function in *TYP* because it contributes unpleasant affect to *Q*. In more detail:

Q - A representation or an awareness of (in the veridical case) the noxious intensity and bodily location of thermal and mechanical energy.

U - A mental state which functions to motivate its subject in whatever injury-preventing way is relevant to the content of *Q* by making it feel as if *Q* were intrinsically unpleasant.

Unlike perceptualism and Klein's motivationalism, the mixed position conforms naturally to the structure of *FS*. Consider my looking at strawberries in a bowl. I see them *and* have an urge to eat them. Although the detail of what it takes to *see* the strawberry is likely to be controversial, almost everyone can agree that my visual experience is perceptual and that my urge is motivational. So there should be no problem with the idea that there is an intimate connection between perceptual and motivational mental states. Indeed, we might say that in this case, visual phenomenology and an urge are constituents of a compound (or composite) perceptual/motivational experience. Of course, a lot more needs to be said about the

role that such factors as concepts, memories and non-cognitive mechanisms (involved in satiation, for example) play in linking perceptual and motivational mental states, but the parallel with *TYP* as conceived by mixed theorists is clear. By the mixed theory, *Q* is a perceptual mental state with (non-conceptual) content about noxious energy, which is linked to a motivational mental state (*U*). Perceptualism treats *U* and Klein's motivationalism treats *Q* in ways that exaggerate their asymmetry with visual experiences (say), because the former takes *U* to be perceptual rather than motivational like the urge in the visual example above and the latter takes *Q* to be motivational and not like the perceptual experience of seeing strawberries.

Background conditions like concepts, memory and non-cognitive mechanisms that are involved in the linking of visual experiences with motivational mental states sound like the modulatory factors that scientists posit as an explanation for the variable relationship between pain and the stimulus (noxious intensities of energy detected by sensory receptors).[72] In the visual case, we do not expect there to be a one to one relationship between a visual experience and a motivational mental state; at one time my urge to eat the strawberries will be greater than another and at some other time I may lack the urge to eat the strawberries. The same might be said of *TYP*. We do not distinguish between the dissociable components of *TYP* in our everyday concepts. Likewise, most of the research on the factors that modulate *TYP* does not distinguish between influences on *Q* and influences on *U*. But armed with the knowledge that *TYP* is a composite experience and the broad conception of a nocifensive functional system (*FS*), the mixed theorist can parallel the variable relationship between visual

[72] I do not discuss modulatory factors in any detail at any point in this thesis even though they are mentioned at various points. For a little more detail see chapter 3 section *3.1*.

phenomenology and motivational mental states with the variable relationship between *Q* and *U*.[73] There is at least some evidence that the opioids that mediate some modulatory influences on *TYP* have a greater effect on the experience of *U* than *Q* (Van der Kam, et al, 2008). So there are evidential grounds for the claim that *Q* fairly accurately corresponds with the occurrence and noxious intensity of energy at a bodily location. In cases of noxious intensities of energy in the absence of pain (P5), it is open to mixed theorists to claim that *Q* most often accurately represents the stimulus, but subjects fail to notice *Q* because it is not experienced in conjunction with *U*. If *Q* was experienced as if it were intrinsically unpleasant it would grab the subject's attention. The absence of *U* in such circumstances is explained by modulatory factors such as the perceived context. However, it cannot explain *Sprain* (see above section *2.1.2* and chapter 1, section *1.2.5*).

By *Sprain* there is a variable relationship between the intensity of *TYP* and the intensity of the mechanical energy being imposed on the subject's ankle. Although, it is not clear how much the respective intensities of *Q* and *U* are contributing to the variable intensities of *TYP*, it is explicit that the subject experiences *TYP* from the moment she begins to rise. Now if it is stipulated that the intensity of energy being imposed on her ankle passes the noxious threshold when she is imposing moderate weight on it (at three minutes, say) then it is clear that for the first three minutes the subject's experience, which is partly constituted by *Q*, is non-veridical. This is a problem for the mixed position. Moreover, *TYP* is decreasing in intensity throughout the full six minutes, while the mechanical energy being imposed on the subject's

[73] The variable relationship between the intensities of *Q* and *U* is part of P6 (see chapter 1, section *1.2.5*).

ankle is increasing in intensity. This suggests that *TYP* is misrepresenting the noxious energy throughout the six minute period.

In response, it might be tempting to claim, contrary to appearances, that *Q* is increasing in intensity in parallel with the magnitude of mechanical energy from the three minute point. The impression that the intensity of *TYP* is moderating is then explained by a decrease in the intensity of *U* – I am assuming here that a *TYP* constituted by a high intensity token of *Q* and a low intensity of *U* will feel less intense than a *TYP* constituted by a low intensity token of *Q* and a high intensity of *U*.

This strategy is unsatisfactory for two reasons. First, *Q* has an intensity during the first three minutes in which it is non-veridically representing the energy. It is reasonable to assume then that as the intensity of the energy increases towards the noxious threshold that the intensity of *Q* is also increasing. So when the noxious threshold of the tissue is reached, when the energy is least noxious, the intensity of *Q* will be non-veridically representing the noxious intensity of the energy. It is also reasonable to assume that the intensity of *Q* continues to increase as the noxious intensity of energy increases. For these reasons, the disparity between the intensities of noxious energy and *Q* would persist throughout the second three minute period. Second, in most circumstances noxiousness and the need to motivate would be expected to be roughly proportional to one another; if energy is being represented as increasingly noxious, then the intensity of the motivational experience should also increase. But this strategy posits an inverse relationship between *Q* and *U*. This seems implausible.

It is hard to see how the mixed position can cope with the problem posed by *Sprain* without conceding that these experiences misrepresent. Even if the stipulation is changed so that the intensity of energy being imposed on her ankle is noxious from the moment the subject puts her foot to the floor and throughout the six minutes she is upright, the inverse relationship between the intensities of *TYP* and mechanical energy remains a difficult explanatory problem. The trouble is that episodes like this are normal during recovery from injury so it is difficult to bite the bullet and concede that these *TYP*s are non-veridical because it exposes the mixed theory to a great deal of error. Indeed, the importance for the subject of preventing further damage to tissue and the normality of experiences like these strongly suggests that this inverse relationship is adaptive.[74] Nevertheless, given the explanatory advantages of the mixed position I do not think this objection alone is too serious. There is however a functional problem which threatens to undermine the mixed position to a greater degree.

According to the mixed position the content that energy is noxious (the content of *Q*) plays a necessary function in the overall function of experiences constituted by *Q* and *U* (*TYP*). In other words, injury-preventing behaviour is motivated because the *content* of *Q* affects us (*U* provides the affect). But it seems more natural to say that a subject is motivated by *Q because Q* feels unpleasant. If this is right, content plays no functional role in motivating the subject.

[74] I argue for this in chapter 6, section *6.3.3*.

A perceptualist about *Q* can respond by saying that a token of *Q* is identical to its content, i.e. subjectively, there is no distinction to be made between the representational vehicle and content. Consider, experiencing the location of a mechanical sensation like touch. It is very difficult to conceive of a sensation like this that lacks the quality of location. So content about location cannot be dissociated from the phenomenology of touch. Therefore, if *Q* is a factor in the motivation of its subject, then the content of *Q* is a factor in motivating the subject. The problem is that we are not motivated by *Q*. We are motivated by the combination of *Q* and *U* (by *TYP*). *TYP* is the focus of our motivational drive. The motivation is satisfied if *TYP* is resolved or ameliorated to a subjectively acceptable level. We have two very good reasons to prefer this content-less explanation. First, doubts about the veridicality of *Q* fade because the content of *Q* is not a factor in motivation. Second, the content-less explanation is simpler. In short, the motivation of injury-preventing behaviour is amenable to explanation without invoking content.[75] By contrast, perceptual content plays an essential role in the explanation of the function of visual experience. A subject is motivated to eat strawberries because her visual experience has content about strawberries.[76]

Now in response the mixed theorist might concede that the function of *TYP* can be adequately and parsimoniously *explained* without calling on perceptual content, but it

[75] It is difficult to argue that the felt location of *TYP* fulfils no function in motivating its subject so I weaken this stance in chapter 5. In addition I want to make it clear that the claim that perceptual content is not required to explain how a *conscious experience* (of *TYP*) motivates injury-preventing behaviour does not amount to the claim that content about the threat posed by intensities of energy plays no part in *TYP*. My account invokes such content but as a means of explaining a *sub-personal* evaluative process. (See chapter 6, section *6.6.2*)

[76] More precisely, the subject is motivated to eat strawberries *partly* because of her visual experience. Background conditions also play an explanatory role, e.g. the subject's liking for strawberries. However, this detail is unimportant here. The key point is that the content of the experience is a crucial part of an explanation of how visual experience is involved in motivating behaviour.

remains the case that *Q* represents noxious intensities of energy because this position is supported by science. *TYP* is subserved by the nociceptive system, the system that is specifically adapted to detect and transmit information about noxious stimuli. This information is transmitted to the primary and secondary somatosensory cortices, the brain areas where *perceptual* information about tactile stimuli are processed, and which are thought to play a necessary role in evoking *Q*. So there are very good reasons to accept that *Q* has perceptual content despite its *apparent* explanatory redundancy. A mixed theorist might press the point by emphasising that my objections place all the functional burden on *U*. It is barely plausible, given the tight connection between *Q* and *U*, that *Q* fulfils no conscious function. If not a perceptual function what might that function be given the perceptual features of *Q* and the difficulty construing *Q* in motivational terms.

In summary, the mixed position has considerable explanatory advantages over perceptualism and Klein's motivationalism. In particular it handles both *Q* and *U* in a natural way; *Q* appears to be perceptual in nature because it is perceptual in nature, and *U* appears to be motivational in nature because it is motivational in nature. This provides a straightforward explanation for P1 and P2. Furthermore, it can handle many cases where it is reported that *TYP* has not been experienced despite the occurrence of tissue damage (P5) and some cases of the variable relationship between the intensities of *TYP* and noxious intensities of energy (P6). Additionally the mixed position is consistent with the science. It has failings though. It cannot explain cases of *TYP* in the absence of noxious stimuli (P4) and cases like *Sprain*. It is also difficult to explain what role the content of *Q* plays in the injury-preventing function of *TYP*.

So although it does not offer a complete explanation for P1-P6 for all that has been discussed it can be said that the mixed position offers the prospect of a complete solution to P1-P6.

3 THE CONCEPTS OF PAIN SCIENCE

Perceptualism, motivationalism, and the mixed theory are not divorced from pain science. In a simple sense for example, the empirical problems I have presented under P4-P6 could be construed as data. If a theory cannot explain this data then there is a problem with the theory.[1] Pain science deals in data of one sort or another. Its conceptual models frame and are framed by data. Given these resources it is reasonable to assume that pain science is best placed to provide a conceptual model that has the power to explain all of P1-P6.

Broadly speaking, the aim of this chapter is to test this assumption. This is a difficult task for two reasons. Firstly, just like philosophical positions on pain, pain science is a broad church. Fortunately, the variations in scientific opinion are far less extreme than those of philosophy so it is possible to sketch out a broad understanding of pain that should be acceptable to most commentators, even if these commentators might disagree about the detail. Secondly, and more crucially, the scientific literature is beset by ambiguities and inconsistencies which give the distinct impression of a lack of development. Although I will attempt to work through these difficulties *where relevant*, this chapter should not be seen as a comprehensive attempt to clarify the multidimensional, pain matrix and nociceptive models I consider here. In the final section of this chapter, I rationalise these interrelated concepts into a single hybrid

[1] This is the methodology I have adopted in this thesis.

which strengthens mixed perceptualism/motivationalism but, I argue, it cannot address all of P1-P6.

This chapter is divided into four sections. The *multidimensional model* – the view that *TYP* has sensory-discriminative, affective-motivational and cognitive-evaluative dimensions – is the focus of section *3.1*. Section *3.2* is divided into two sub-sections. In the first I introduce the pain matrix, which concerns the functional anatomy of the brain. The pain matrix is an anatomical and functional map of the brain areas involved in pain. It links the dimensions of the multidimensional model with the peripheral neurology of the nociceptive system. In the second sub-section, I criticise the specificity implied by the pain matrix. In section *3.3*, I present a simple introductory account of the nociceptive system and gesture towards some of the ambiguities that will be my focus in chapter 4. In *3.4*, I develop a hybrid of these models and argue that it cannot solve the explanatory problems expressed in P1-P6.

3.1 THE MULTIDIMENSIONAL MODEL OF PAIN

The multidimensional model of pain[2] is "so widely accepted as established fact and is such a central tenet of pain research that most people are unaware that it is only an hypothesis and, moreover, one that has never been seriously tested" (Fields, 1999, p.S65). Part of the problem, and it is one that Fields highlights in his account, is that it is far from clear what it means to say that pain has sensory-discriminative, affective-

[2] Although the concept that pain has sensory-discriminative, affective-motivational and cognitive-evaluative dimensions is so widely accepted amongst pain scientists, the *term* 'multidimensional model' is rarely if ever used. To my mind it is a wholly acceptable term.

motivational and cognitive-evaluative dimensions. Fields' concern is partly that the headline pairings of 'sensory' with 'discriminative' and 'affective' with 'motivational' are ambiguous and from different conceptual domains respectively (1999, p.S64-S65). This section is devoted to unpicking these ambiguities.

What is not in doubt, is that the 'pain' of the multidimensional model refers to *TYP* (i.e., to the experiences, which are constituted by *Q* and *U*, that we typically identify as pain). Because of ambiguities surrounding the constitution of pain that I outlined in chapter 1 section *1.2.3*, I will continue to refer to these composite experiences as *TYP*. So the question is whether the multidimensional model should be viewed narrowly as the concept that *TYP* is *constituted by* sensory-discriminative, affective-motivational and cognitive-evaluative components or whether a broader reading, by which *TYP* *involves* sensory-discriminative, affective-motivational and cognitive-evaluative dimensions, is more appropriate.

Putting detail aside, a broad reading is rather trivial.[3] It is clear that *TYP* is *influenced by* multiple factors including sensory input, attention, expectation, memory and context and that *TYP* has the capacity to affect emotions, memory and to motivate behaviour.[4] These factors can be classified as sensory, affective and cognitive. There is also powerful evidence that *TYP* is *constituted by* two components.[5] This much has become basic pain science. The narrow reading is more interesting, but doubts about

[3] The details are not trivial.
[4] See Melzack (1999) and Tracey and Mantyh (2007) for more on the multiple influences on and of *TYP*.
[5] See chapter 1, section *1.2.2*.

what a sensory-discriminative and in particular a cognitive-evaluative dimension amount to favour the broad understanding.

In chapter 1 section *1.2.2*, I presented evidence that *TYP* is constituted by two mental states, an affectively neutral phenomenal quality which I have labelled '*Q*' and a mental state '*U*' which makes it feel as if *Q* is intrinsically unpleasant. This understanding reflects the composite nature of *TYP*. By the narrow reading, the multidimensional model *TYP* is *constituted by* sensory-discriminative, affective-motivational and cognitive-evaluative dimensions. Although, the psychofunctional structure of the label 'affective-motivational' worries Fields (1999, p.S64) it is easily accommodated by the composite structure of *TYP*; the affective nature of *TYP* functions to motivate its subject. To my mind this is nothing more than an acknowledgment that the affective component of *TYP* (presumably this component is *U*) has a motivational function. Given the evidence I presented in chapter 1, section *1.2.4* this much should not be controversial.

The sensory-discriminative component is not so amenable to a psychofunctional treatment for a couple of reasons. Firstly, 'sensory' is a functional term that implies discrimination. For example, nociceptors (the sensory receptors that detect noxious intensities of energy) have a sensory function, they discriminate noxious stimuli. By this understanding of the sensory-discriminative component 'sensory' and 'discriminative' are both functional terms, and unlike 'affective' and 'motivational' neither term obviously refers to the subjective nature of *TYP*. Now it would be easy to be charitable and either pass this off as a terminological matter ('sensory' should be

taken to refer to the affectively neutral phenomenal qualities of *TYP*, and 'discriminative' to the function of these qualities) or to ignore the issue altogether if it were not for P4-P6. The trouble is that *TYP* (and *Q*) are not closely correlated with the noxious stimuli. The question is how can a mental state function as a conscious representation or awareness of something if the experience is unreliable in that role. I will put this question to one side for a moment because the solution, which is prompted by consideration of the cognitive-evaluative dimension, is that the multidimensional model is best interpreted in broad terms.

If the above problem is ignored the sensory-discriminative and affective-motivational dimensions can be neatly accommodated into a narrow reading of the multidimensional model. *TYP* is exhausted by two subjective components *Q* and *U*. The affectively neutral phenomenal qualities of *Q* have a sensory-discriminative function and the unpleasant affect of *U* motivates injury-preventing behaviour. A cognitive-evaluative component complicates this neat structural picture because sensory and motivational functions have already been ascribed to *Q* and *U* respectively. On the face of it this is not a significant worry, either *Q* or *U* or both *Q* and *U* are intrinsically cognitive-evaluative as well as sensory-discriminative and affective-motivational. But what does it mean to say that *TYP is* intrinsically cognitive-evaluative?[6]

[6] Fields sees what he calls 'secondary unpleasantness' (as opposed to primary unpleasantness) as the locus for the cognitive-evaluative dimension (1999). It is far from clear what he means by this term. Donald Price writes of 'secondary affect', by which he means that *TYP* has an effect on various factors including cognition and evaluation that is particularly marked when *TYP* is prolonged (2000). Perhaps Fields means that the unpleasantness of *TYP* (primary unpleasantness?) affects cognitive-evaluative processing and this in turn has a negative (secondary) affect on primary unpleasantness.

Cognition and evaluation are certainly involved in *TYP*. Consider my evaluation that today's visit to the dentist will be just like my previous visit. This evaluation has causally contributed to my current anxiety, and this in turn has the potential to adversely affect the intensity of my experience of *TYP* at my coming appointment.[7] These aspects of *TYP* are bi-directional. The *TYP* I experienced when I first visited the dentist had a primary affect on cognitive-evaluative processing, and cognitive-evaluative processing has a secondary affect on my subsequent experience. In more detail: I have a memory of my initial visit to the dentist, which includes memories of *TYP* and the circumstances in which it occurred. I have also evaluated today's visit in the light of this memory, and concluded that it is likely to be similar to my previous visit. This makes me anxious, and has the capacity to have a causal affect on *TYP* at my coming visit to the dentist.[8] However, I do not think that this sort of example provides a strong case for the narrow reading.

Clearly, *TYP* affects cognitive-evaluative processing and *vice versa*, but the key question is whether this is an intrinsic function of *TYP*. Here is a reason to think it might be. An experience of *TYP* has the capacity to motivate future behaviour via cognitive-evaluative processing. In the example above, my anxiety functions to motivate me *not* to repeat a markedly unpleasant experience. This anxiety would motivate me accordingly if it were not for other cognitive-evaluative considerations; in particular, an understanding that my dental health will benefit from the second visit.

[7] The precise mechanism of this effect is uncertain, but there is evidence that it involves the blocking of inhibitory mechanisms with an antagonist substance called cholecystokinin (Benedetti, et al, 2007).
[8] With respect to the affect of anxiety on *TYP*, the evidence is not entirely clear. Some commentators report that anxiety increases the unpleasantness of *TYP* (e.g. Horn et al, 2012) while others report that it has little or no effect (e.g. Arntz et al, 1994). It may be that attention is the key factor, i.e. that anxiety focuses the subject's attention and it is this focus which determines whether *TYP* is more unpleasant. If this is right anxiety is a causal factor in some pain experiences. I am taking this to be the case here. All this goes to show how complex the topic of modulation is. (See Moseley, 2007, for further comment.)

The point is that *TYP* is affecting cognitive-evaluative processing and this has the potential to affect advantageously my future behaviour. As this cognitive-evaluative function is an aspect of injury-preventing behaviour and the motivation of injury-preventing behaviour is the intrinsic function of *TYP*, *TYP* is partly constituted by a cognitive-evaluative dimension.

It might be wondered what it means to say that a mental state has an *intrinsic* function, but this would be a diversion at this stage.[9] To my mind matters are settled against the narrow reading by consideration that *TYP* is constituted by affective phenomenology; i.e. by an affectively neutral phenomenal quality (*Q*) and a mental state (*U*) that makes it feel as if *Q* were intrinsically unpleasant so that we are affected by the composite (*TYP*). These intrinsic features of *TYP* have an effect on cognition and evaluation and *vice versa*, but cognition and evaluation are not constituents of *TYP* in this intrinsic sense, even though it can be argued that *TYP* has (in part) the function of affecting cognitive-evaluative processing.

As I understand it, the multidimensional model should be interpreted as a broad model by which *TYP* has sensory-discriminative, affective-motivational and cognitive-evaluative *dimensions*. These encompass functional influences *on TYP*, as well as the functions *of TYP*. The sensory-discriminative and affective-motivational *components*, which constitute *TYP*, are aspects of the respective dimensions. Hence, *TYP* has a cognitive-evaluative dimension, but it lacks a cognitive-evaluative component.

[9] I address this issue in chapter 5, section *5.1*.

In the earlier part of this section, I said that a broad reading of the multidimensional model is rather trivial. However, this statement is only true as far as the general statement that pain is multidimensional is concerned. The specifics of the dimensions of this model are far from trivial. As far as my thesis is concerned, the importance of the broad reading is that the sensory-discriminative function attributed to *TYP* under the narrow reading is not a consequence of the broad reading. *TYP* could lack a sensory-discriminative component but have a sensory-discriminative dimension where sensory discrimination is involved in *TYP*, but is not represented by *TYP*. So the position that both *Q* and *U* have motivational functions is not contradicted by a broad reading of the multidimensional model.

In summary, the multidimensional model conveys an awkward mix of function and intrinsic nature. According to this model, *TYP* has sensory, motivational and cognitive functions. *TYP* has the capacity to function in this way because it has sensory qualities (varied phenomenal qualities, bodily location and intensity) and it is affective. The affect can motivate both directly and indirectly. The latter involves cognition and evaluation. I take it that *Q* corresponds with the sensory-discriminative *component* and *U* with the affective-motivational *component*. Although, on balance I am unsure whether the three dimensions of the multidimensional model are intended to be interpreted broadly, this reading of the *dimensions* of *TYP* is most sympathetic because it is consistent with the position that the cognitive-evaluative dimension is not a constituent of *TYP*.

As it stands, the multidimensional model is not equipped to address P1-P6. It does not explicitly distinguish *Q* and *U*, but even if it is interpreted, as I have done here, in terms of *Q* and *U* it has nothing to say about content or how the sensory and motivational components combine to yield injury-preventing behaviour. Although the cognitive-evaluative dimension provides a means of explaining at least some of the problems posed by the weak correlation between pain and the stimulus (P4-P6) this dimension needs filling out. The following sections on the pain matrix and the nociceptive system can be seen as adjuncts to the multidimensional model that increase its explanatory power.

3.2 THE PAIN MATRIX

Although the 'multidimensional model' and 'pain matrix' are distinct conceptual models there is a strong link between the two as activity in the brain areas of the pain matrix is considered to be the neurological substrate of various aspects of the multidimensionality of pain, including *TYP* itself. In short, the pain matrix lists the functionally and anatomically discrete brain areas that are considered to be involved in *TYP*. This may sound simple, but it conceals significant neurological and conceptual complication, much of which is beyond the scope of my thesis.

The pain matrix is a development of Melzack and Casey's '*neuromatrix*' concept. By the latter, pain is an output of a network of brain areas that are not specific to pain (1968). These brain areas integrate inputs from a variety of sources, including nociceptive and non-nociceptive sensory inputs (Melzack, 1999). Recently, Ianetti

and Mouraux have expressed concerns about this development. They claim that advocates of the pain matrix are assuming that the brain areas of the pain matrix are specific to pain (Ianetti and Mouraux, 2010; Mouraux et al. 2011).[10] Despite the specificity of the pain matrix, I am certain that advocates of the pain matrix like Tracey and Mantyh, and Treede and colleagues accept the overwhelming evidence that the brain areas of the pain matrix fulfil multiple functional roles, which are not limited to pain.[11] There is consensus on this matter; all accept that the brain areas of the pain matrix are *not* specific to pain. My understanding is that the concept of a pain matrix is a means of framing the burgeoning understanding of the brain areas that are involved in pain. Nevertheless, the pain matrix is misleading. It is not just a re-labelling of Melzack and Casey's 'neuromatrix'.[12] This matter is of particular importance because I use a conceptual model as a means of framing the various aspects of my thesis. This framework (the nocifensive functional system – *FS*) is much more like Melzack and Casey's neuromatrix than the pain matrix.

This section is divided into two sections. In the first, I provide a simple account of the pain matrix and in the second, I criticise and develop this account.

[10] If Ianetti and Mouraux were correct it would suggest that scientists widely accept that we are close to being able to *see* pain by reading pictorial representations of scanning data. This would have important legal implications. (See Camporesi, et al, 2011.)

[11] Treede et al explicitly refer to the multiple functional roles of various brain areas of the pain matrix. These functions are not specific to pain (1999, p.109, Table 1), which refers to the multiple functional roles of various brain areas of the pain matrix. See also Medford and Critchley for a discussion of the diverse functions of two areas of the pain matrix, the anterior cingulate cortex and the anterior insula cortex (2010), and Seward and Seward who present an account of the role that the thalamus and periaquaductal grey may play in pain. The latter make it clear that the functions of these brain areas are not restricted to pain (2002).

[12] Tracey and Mantyh seem to be making this claim. They write that Melzack and Casey first referred to the network of brain areas that is involved in *TYP* as "the pain 'neuromatrix', but it's now more commonly referred to as the 'pain matrix'" (Tracey and Mantyh, 2007, p.379).

3.2.1 The specificity of the pain matrix

In its simplest form the pain matrix is a map of the brain areas involved in *TYP*, their connections and functions. There is debate about which brain areas constitute the pain matrix and about the functional role that each brain area plays in *TYP* (Ianetti and Mouraux, 2011; Tracey and Mantyh, 2007)). This understanding implies modularity, the concept that the brain is divided into anatomically distinct regions with discrete functions. This assumption is at least an oversimplification.[13] Given modularity, part of the difficulty with the pain matrix is that brain areas which are distinguished anatomically are associated with varied functions. For example, the anterior cingulate cortex, which has been identified as a key component of the pain matrix, has been associated with executive function, error detection, motivational valence, and other functions (Medford and Critchley, 2010). For this reason, theorists should be cautious about taking putative detail concerning the discrete function of a brain area as unequivocal support for a particular position. Rather it is reasonable to claim that the attribution of a specific functional role to a brain area adds to the strength of the case for a position. In this light, the pain matrix concept provides some support for the mixed theory. In particular, the widely held view that activity in the primary and secondary somatosensory cortices (SI and SII) is associated with both *Q* and sensory-discriminative function, and activity in the anterior cingulate and insula cortices (ACC and IC) is associated with both *U* and motivational function.

[13] See Liang et al (2013) for evidence that brain areas which are normally assumed to be silent in a given experiment may be contributing in some relevant way. See also Hardcastle and Stewart (2002), for an account of the limitations and assumptions made in the collection and interpretation of brain data.

SI and SII receive nociceptive inputs via the spinothalamic tract (STT), while ACC and IC receive nociceptive inputs via STT, the spinoreticular (SRT) and spinomesencephalic tracts (SMT) (Tracey and Mantyh, 2007, p.378; Willis et al, 2002, p.90). Together the tract and brain areas of the former are sometimes conceived as the *lateral pain system* and the tracts and brain areas of the latter are the *medial pain system* (Scherder et al, 2003, p.p.678-679).[14] As the brain areas associated with both the sensory-discriminative and affective-motivational dimensions of *TYP* (SI and SII, and ACC and IC respectively) receive nociceptive input via parallel pathways it might be claimed that SI, SII, ACC and IC process sensory input. But despite this link scientists do not associate ACC and IC with a sensory-discriminative function.[15] This supports the understanding I expressed in the preceding section that the terms 'sensory-discriminative', and 'affective-motivational' refer to functional outputs; i.e. SI, SII, ACC and IC all process sensory input; SI and SII are sensory-discriminative because they yield perceptual outputs (including *Q*) and ACC and IC are affective-motivational because they yield motivational outputs (including *U*).

In chapter 2 section *2.1.5*, I briefly introduced the concept of a nocifensive functional system (*FS*) as a means of understanding the connections between sensory input and outputs (including *TYP*) that have the potential to yield nocifensive behaviours. On this understanding both the affectively neutral phenomenology (*Q*) and the affective

[14] I am simplifying matters a great deal here. Scherder et al also associate the ACC with cognitive-evaluative and memory functions, and the IC and SII with a memory function (2003). So it is extremely difficult to make unequivocal functional claims about areas of the pain matrix. This detail does not make a difference to my case for motivationalism nor is it troublesome for the mixed position or perceptualism, but it does show that philosophers should be very careful about claiming that science provides unequivocal support for a position.

[15] This is a massive oversimplification of the medial and lateral pain systems. In particular the medial pain system is associated with various thalamic and hypothalamic nuclei, the locus coeruleus, the periaquaductal grey, the parietal operculum, the hippocampus and the amygdale. The thalamus is also a constituent of the lateral pain system (Scherder et al, 2003).

mental state (*U*) that constitute *TYP* fulfil functional roles in a system that has the potential to yield injury-preventing behaviour. This is consistent with the view that sensory input from the nociceptive system fulfils a very important role in an affective mental state that has a motivational function. On this view, the ACC and IC are involved in the processing of sensory input and they are (necessary?) elements in the evocation of *U*. Hence, ACC and IC have sensory (the processing of nociceptive input) and motivational (they play a necessary role in *U*) functions, while *U* has a motivational function but it is not sensory.[16] *FS* is also consistent with the view that sensory input fulfils a very important role in phenomenal qualities that fulfil a perceptual function. On this view, SI and SII function to process sensory input and they are (necessary?) elements in the evocation of *Q*, which has a perceptual function. But note here that the processing of nociceptive input by SI and SII, and by ACC and IC does not imply that *Q* and *U* have perceptual and motivational functions respectively. ACC and IC might be involved in evoking a mental state that has a perceptual function[17] and SI and SII might yield a mental state that has a motivational function. What counts against the former is the evidence that those with asymbolia are not motivated by *Q* and the evidence that *Q* is affectively neutral counts against the latter. Functional anatomy merely adds weight to the subjective evidence that the composite experience *TYP* has a motivational function and that *U* is at least a necessary constituent of that function.

[16] ACC and IC are not normally associated with the processing of sensory input. My thinking is that as ACC and IC receive sensory input and yield various outputs then they *must* process (or be involved in the processing of) the input.

[17] Scherder, et al, link IC with affective-motivational and *sensory-discriminative* functions and SII with sensory-discriminative and *affective-motivational* functions (2003).

The concept of medial and lateral pain systems links the nociceptive system with the functional anatomy of the pain matrix. In the next sub-section I will explain why this is an awkward association, but here I want to point out that the evidence of connections between nociceptive neurons and various brain areas of the pain matrix is evidence that the relevant brain areas have a sensory-discriminative function. In places I have referred to this function as the '*processing* of nociceptive (or sensory) input'. My use of this term is largely consistent with its wide use in the scientific literature.[18] I say largely, because it is not entirely clear what scientists mean by 'processing' in the context of the pain matrix. It seems that there is some disparity between my broad understanding and that of others. Contrary to current opinion, I take it that ACC and IC are involved in nociceptive processing on the grounds that they receive nociceptive input. This is sensory processing in the sense that activity in sensory receptors is the catalyst for the transmission of action potentials in afferent (sensory) pathways to ACC and IC.

3.2.2 A broad understanding of the pain matrix

To recap, the concept of the pain matrix has recently faced some opposition.[19] On a conventional and simple understanding, the pain matrix is constituted by the network of brain areas that are involved in *TYP*. Additionally, these brain areas are ascribed a (putative) functional role in *TYP*.[20] For example ACC is conceived as a constituent of the pain matrix that has affective-motivational, cognitive-evaluative and memory

[18] For example, see, Ohara et al, (2005); Perl (1998); and Tracey and Mantyh (2007).
[19] By Ianetti and Mouraux (2011) and Mouraux, et al. (2011).
[20] See section *3.2*.

functions for *TYP*. But this apparently uncontroversial conception is in tension with the knowledge base of pain science because it strongly suggests an unjustified specificity. Inadequate expression is a part of the problem. For example, pain scientists often use terms like 'painful stimulus', 'peripheral pain-signalling-neurons', 'pain pathway' and 'pain impulses', which suggest the view that the nociceptive system is specific to *TYP*. However, I very much doubt that any pain scientist holds such a view because the overwhelming evidence is that the nociceptive system is not specific to *TYP*. For example, it mediates inflammatory responses and provides input to the motor system. This tension is evident in Tracey and Mantyh's summary of the relationship between the multidimensionality of *TYP*, the brain and sensory input: "Because pain is a complex, multifactorial subjective experience, a large distributed brain network is subsequently accessed during nociceptive processing" (2007, p379). The important thing to note is that the multidimensionality of *TYP* is being given as a reason for positing the pain matrix, but the brain areas of the *pain* matrix are linked with *nociceptive* processing. This would not be of any consequence if *TYP* was closely correlated with nociceptive stimuli, but the evidence is that it is not (P4-P6). This evidence has prompted the IASP to explicitly "avoid tying pain to the stimulus" (2014).

Tracey and Mantyh's comment can be interpreted in the light of the IASP's statement that "pain most often has a proximate physical cause" (2014). By this, the IASP mean that *TYP* is *normally* causally related to the detection of a noxious stimulus. But even if *all* tokens of *TYP* were causally related to such a stimulus, it would be a mistake to assume that correlations between activity in a 'large distributed brain network' and

both *TYP* and noxious stimuli warrants the claim that all the activity in this network is pain-related. This is because the nociceptive neurons that are presumed to have detected these stimuli are involved in responses other than pain. Hence, if the pain matrix is taken to be constituted by the brain areas which are determined to be active by scanning data or by the "projection pathways originating in the nociceptive areas of the spinal cord dorsal horn" (Treede, et al, 1999, p.106)[21] the label 'pain matrix' is at best misleading. The label 'neuromatrix' or ('nociceptive neuromatrix') would be more appropriate. At worst it reflects harmful confusion that reflects the unsupportable specificity evident in inappropriate terms like 'pain pathway'.[22] What then is the pain matrix?

There is little doubting the utility of the notion that a network of brain areas with various functions is involved in *TYP*, and that different patterns of activity in this network (different '*neurosignatures*'[23]) are causally involved in different tokens of *TYP*. Medically, it opens practitioners eyes to the multiple causal influences on the tokens of *TYP* that they see every day in their practice, and theoretically it reflects our current understanding of the functional contribution of different brain areas to *TYP*. But it is evident from i-v below that packaging the science into a discrete conceptual model is a gross oversimplification.

[21] This is a method of tracing the anatomical connections of nociceptive neurons.
[22] It is harmful in a very real sense. The notion of pain specific pathways has been a factor in the inadequate treatment of chronic pain. This has been one of the major motivators of modern pain science. In particular, it has been a significant factor in the IASP's decision to define pain as a subjective experience and to dissociate pain from the stimulus. So it is surprising that many pain scientists are so imprecise in their expression. (See Siddall and Cousins, 2004; Merskey, 1986; and the IASP's Taxonomy on Pain, 2014).
[23] This term is not mine. Michael Thacker was using it in 2007 at King's College, London. David Butler and Lorimer Moseley have used the term 'neurotag' in various settings and Tracey and Mantyh refer to a 'pain signature' (2007).

Given that activity in the brain areas which constitute the pain matrix is not necessarily specific to *TYP*, and that activity in the brain which is *TYP*-related may not yield *TYP* consider a brain area ('*X*') that is sometimes involved in *TYP*-related activity. The following illustrates some possibilities:

i) Activity in *X* is *TYP*-related and a token of *TYP* results from all the relevant (*TYP*-related) brain activity.

ii) Activity in *X* is *TYP*-related and no token of *TYP* results from all the relevant brain activity.

iii) Activity in *X* is not *TYP*-related and a token of *TYP* results from all the relevant brain activity.

iv) Activity in *X* is not *TYP*-related and no token of *TYP* results from all the relevant brain activity.

v) Activity in *X* is not *TYP*-related and all of the brain areas that are sometimes involved in *TYP*-related activity are involved in activity that is not *TYP*-related.[24]

The pain matrix cannot adequately capture what is going on in i-v. On a conventional understanding *X* is clearly a constituent of the pain matrix because it is causally involved in some tokens of *TYP*. But as activity in *X* is not necessarily *TYP*-related, then it is causally involved in other matrices as well. So we might say that *X* is also a constituent of the immune response matrix and the motor matrix, say. It is clear from this that the conventional understanding favours anatomy over physiology and

[24] It is worth adding that the list of possibilities would have been further complicated if I had also considered the fact that activity in brain areas of the pain matrix is not specific to nociceptive input. However, i-v are sufficient for my purposes.

function. A *brain area* is a constituent of the pain matrix just because it has the capacity for *TYP*-related activity. This is uninformative because it says nothing about the function of token activity in a brain area. In my view, it is a mistake to think of a brain *area* as a constituent of the pain matrix, what matters is whether activity in a brain area is *TYP*-related. By '*TYP*-related activity' I do *not* mean activity that has the *potential* to influence *TYP* because there are good reasons to think that such activity is going on most if not all of the time.[25] For example, cognitive processing linked to the evaluation of its subject's environmental context is likely to be a significant feature of that subject's mental life and it always *has the potential* to be a factor in a token of *TYP*. A broad understanding like this would not illuminate any of i-v unless it could be explained why some activity in a brain area of the pain matrix had no potential to influence *TYP*. In the absence of such an explanation it would be reasonable to assume that all activity in *X* could influence *TYP*. So '*TYP*-related activity' refers to activity that *is* influencing *TYP*. To be clear on this *TYP*-related activity might yield *TYP* (i), or it might inhibit so that *TYP* is not experienced (ii).

In this light, we can say the following about some particular activity (Φ) in a given brain area (*X*) that has the potential to influence *FS* at various times t_1- t_4:

a) At t_1, Φ in *X* is functioning solely as a constituent of *FS*.

b) At t_2, Φ in *X* is functioning as a constituent of *FS* as well as some other functional system(s).

[25] If *TYP*-related activity was defined as the potential to influence *TYP* and some *TYP*-related activity was going on all the time, it would contradict v.

c) At t_3, Φ in *X* is functioning as a constituent of *FS*, while other activity in *X* (*Σ*) is functioning as a constituent of another system.

d) At t_4, Φ in *X* is not functioning as a constituent of *FS*.

Note that Φ in *X* has the potential to be *TYP*-related activity but is not necessarily *TYP*-related activity (it is *TYP*-related activity at t_1-t_3 but not at t_4), and that *X* yields different activity (*Σ*). By *a*-*d* it is possible that *Σ* in *X* has the potential to be *TYP*-related. Note also that, unlike i-v above, *a*-*d* make no mention of *TYP*. It is an open question whether or not there are tokens of *TYP* at t_1-t_4. The implication of this is that activity in *FS* may not evoke *TYP*. Indeed, it may be that at t_1 Φ in *X* is a factor in the inhibition of nociceptive input to the central nervous system. So overall the net state (the 'all things considered' state given by the sum total of the integration of all *TYP*-related activity) of the pain matrix may be described as inhibitory, excitatory or neutral with respect to *TYP*.

I want to emphasise that this is not intended to be an exhaustive analysis. Acceptance of the standard picture that a (any?) given brain area has the potential to fulfil varied functional roles and that physiological activity is necessary for these functions is enough to open up the sorts of possibilities I have set out in i-v and *a*-*d* above. It is obvious from this that a model of *TYP* that focuses on the functional anatomy of brain areas will be inadequate. However, it is one thing to theorise using placeholders like *X*, Φ and *FS*, it is another thing altogether to fill the slots occupied by these placeholders with anatomy, physiology and worked out systems. As things stand the pain matrix represents science's best attempts to fill in *X* (the brain areas that have the

potential to be involved in *TYP*-related activity) and to establish the functions of the *TYP*-related activity of these brain areas. The above analysis just shows how much still needs to be done.

The problem with the literature on the pain matrix is that it fails to place functional anatomy in a wider context that reflects the multiple functions of the *physiological activity* that takes place in these brain areas. In my view Melzack and Casey's conception of a 'body-self neuromatrix' constituted by widely distributed brain areas with varied functions (Melzack 1999) is a better framework for the established functional anatomy. Its great advantage is that it is not conceived to be specific to either *TYP* or nociceptive input so it can accommodate some of the issues raised by i-v and *a-d*. Unfortunately, the neuromatrix lacks a great deal of important conceptual detail. Part of the reason for this is that it has been superseded by the pain matrix. But the pain matrix is not a substitute for the neuromatrix. The functional anatomy that has been framed in terms of a specific pain matrix would be better served by consideration in the broader context of a neuromatrix. Interestingly, scientists who discuss the pain matrix refer to individual neurosignatures and modulatory influences even though these concepts are not readily accommodated by the pain matrix – these concepts are reflected in the issues raised by i-v and *a-d* – so the best *accounts* of the pain matrix look more like accounts of the neuromatrix.

The conceptual framework that I set out in chapter 5 section *5.6* is broadly based. Like the neuromatrix it takes *TYP* to be *an* output of a system that receives multiple inputs. But unlike the neuromatrix, which is grounded in the concept that *TYP* emerges from

a broadly construed and rather intangible 'body-self' that is conceived in terms of both functional systems and neurobiology, my nocifensive functional system (*FS*) is grounded in a specific functional role; the production of nocifensive responses and behaviours. So *FS* is more specific than the neuromatrix and it is less specific than the pain matrix.

These considerations are not comprehensive. My purpose in this section has been twofold. First, to highlight the misleading specificity that arises from failing to place functional anatomy in a broader conceptual framework. A superficial understanding of the pain matrix is likely to lead to the impression that a specific network of brain areas is specifically involved in processing input from the nociceptive system and yielding a particular multidimensional experience (*TYP*) as an output. This impression is not supported by science. Even so my argument should not be seen as a rejection of specificity. In the account I have given above, *TYP*-related activity has a specific causal effect on the net state of the pain matrix. Second, for the purpose of my thesis the anatomical specificity of the pain matrix is not helpful. This is why I develop an explanatory framework (*FS*) that is based on function.

3.3 THE NOCICEPTIVE SYSTEM

The nociceptive system is the missing piece in the neurological jigsaw. The multidimensional concept refers to the functions on and of *TYP*, and the pain matrix and the medial and lateral pain systems provide anatomical, physiological and functional accounts of the CNS substrates of these dimensions of *TYP*. The

nociceptive system concerns the peripheral neurology that provides the sensory input to the pain matrix.

To misappropriate Fields' comment on the multidimensional model of pain,[26] the concept of a nociceptive system is "so widely accepted as established fact and is such a central tenet of pain research that most people are unaware that it is only an hypothesis and, moreover, one that has never been seriously tested" (Fields, 1999, p.S65)." Indeed, most scientists would probably take the specificity, which is explicitly conveyed by nociceptive terms, as being an established fact. In chapter 4, I will deny this specificity, but this is not my purpose here. In this section, I will define and explain the nociceptive system. The IASP's Taxonomy of Pain Terms will provide the raw material for my account. I have done this because the IASP's Taxonomy of Pain Terms represents the *only* attempt to date at achieving a consensus on the meaning of important terms including 'nociception' and 'pain'. Prior to the publication of the IASP's first Taxonomy there was, as John Bonica memorably described, a 'Tower of Babel' situation in which researchers and commentators could not communicate effectively because they often understood key terms rather differently (1979). The fact that the Subcommittee is and has been constituted by many of the foremost thinkers and researchers in the world of pain, like Bonica himself, Martin Koltzenburg, John Loeser, and Harold Merksey and that the Taxonomy is open to frequent review are other good reasons to take the IASP's definitions definitively.

[26] See the opening to section *3.1*, this chapter.

As I discussed in the preceding section, the medial and lateral pain systems are identified with all the peripheral and central structures that are involved in the generation, transmission and processing of nociceptive inputs. I take the nociceptive system to be the peripheral element of these systems. In more detail, it encompasses the structure, processes and connections (the anatomy and physiology) of primary (first order) afferent neurons that are specifically adapted to detect noxious stimuli.[27] The IASP define five 'nociceptive terms' in their Taxonomy, 'nociception', 'nociceptive neuron', 'nociceptor', 'nociceptive pain', and 'nociceptive stimulus' (2014). In this section, I concentrate on the first three of these terms, as detailed analysis of the latter two would add little relevant detail to my account. Crucially, each of the IASP's nociceptive terms is defined, either explicitly or implicitly in terms of a 'noxious stimulus', and the physiological processes of 'transduction' and 'encoding'. I will mention noxious stimuli in this section, but I will not refer to transduction and encoding until the following chapter.

In 1906, Sir Charles Sherrington hypothesised the existence of sensory receptors that are specifically adapted to detect stimuli of a sufficient intensity to threaten the integrity of bodily tissues (Snider and McMahon, 1998). He called these receptors 'nociceptors' after the Latin word *nocere* for harm (Hall, 1989). The implication is that the *specificity* of these receptors is maintained in central structures and is

[27] To a certain extent distinctions like this are arbitrary. The medial and lateral pain systems might be taken to *exclude* primary afferent (nociceptive) neurons. But there would be an air of contradiction about this approach as noxious stimuli, and hence nociceptive activity, is frequently used in experimental design as a means of establishing which tracts and brain areas are involved in these pain systems. As such it seems to more appropriate to label these systems as the 'medial and lateral nociceptive systems'. The distinction between the peripheral and central nervous systems is perhaps a more graphic example of these conceptual ambiguities. The former is constituted by (afferent) primary sensory and (efferent) motor neurons. But both types of neuron have their cell bodies and connections in the central nervous system. Nothing of importance hangs on this issue.

intimately connected with *TYP*. Sherrington's hypothesis was in marked contrast to the opinion that held sway at the time, which was that pain emerges from *a pattern* of brain impulses generated by the activation of non-specific sensory receptors (Woolf and Ma, 2007). The debate between specificity theory and pattern theory has according to some, been settled in favour of the former.[28] However, most commentators are more conciliatory. They recognise that both the specificity of receptors and pathways and patterns of action potentials are important components in the transmission of information about noxious stimuli and experiences of *TYP* (Fields, 2007; Price and Sufka, 2006; Woolf and Ma, 2007).

The frequency of action potentials fulfils an important role in coding (i.e. patterns of impulses that carry coded information) the intensity of a noxious stimulus. But, as far as my thesis is concerned, it is more important to note that the frequency of action potentials is acknowledged as an important factor in distinguishing between noxious and innocuous stimuli. The reason for this is that many of the neurons that are classified as 'nociceptive' begin transmitting action potentials at stimulus intensities that are outside the noxious range (i.e. the stimulus threshold for these neurons is below the noxious threshold for the tissues[29]) (Price and Sufka, 2006). This issue has prompted Bud Craig to suggest that "the category 'nociceptors', while of enormous heuristic value, is actually a theoretical simplification" (2002, p.657).[30] I will discuss

[28] For example, Julius and McCleskey (2006); Perl (1998). Furthermore, the inappropriate use of terms like 'pain pathway(s)' by scientists suggests the widespread acceptance of specificity. Language like this is endemic amongst pain scientists so specific examples are not really needed.
[29] In the preceding chapter, I briefly characterised the 'noxious threshold' as a threshold marking the division between innocuous and threatening intensities of energy. (See chapter 4 section *4.4* and chapter 6 section *6.2.1*, for more on the noxious threshold.)
[30] Craig's suggestion is an extremely rare exception to the wide acceptance of the concept of a nociceptive system.

this matter in more detail in the following chapter, but I want to emphasise that it is a challenge to the notion of specificity.

Doubts about the specificity of nociceptive neurons are not assuaged by the knowledge that nociceptive neurons are a *sub-category* of sensory neuron. Sensory neurons are categorised according to their anatomy as Aα-, Aβ-, Aδ- and C-fibres. Aα-fibres have a relatively thick myelin sheath, 12-22 μm in diameter. As a consequence Aα-fibres conduct action potentials quickly (70-120 ms). Like Aα-fibres, Aβ-fibres are fast conducting fibres (40 ms>) because they have a thick myelin sheath (6-12 μm) (Couteau, et al, 2005; Flor, et al, 2006). Neither Aα- nor Aβ-fibres are categorised as nociceptive. The category 'nociceptive neurons' is constituted by a *sub-population* of the remaining fibre types, Aδ- and C-fibres. Currently there is no principled means of determining which sub-population of Aδ- and C-fibres is nociceptive on the basis of neuroanatomy. All Aδ-nociceptive neurons and Aδ-non-nociceptive neurons are thinly myelinated (1-5 μm in diameter) and they conduct slowly (4-30 ms) as a consequence. Nociceptive and non-nociceptive C-fibres are the slowest conducting fibres of all at 0.4-2ms, because they are unmyelinated (Couteau, et al, 2005; Flor, et al, 2006).

This method of categorisation is focused on an anatomical characteristic of sensory fibres, myelination. It makes no reference to the receptors expressed by these fibres. By contrast, nociceptive Aδ- and C-fibres are distinguished from all other (non-nociceptive) sensory neurons by the stimulus profile of the sensory receptors they express; i.e. by the ability to detect noxious intensities of energy. I will save the

technical detail of the IASP's defintions for the following chapter, but in short a sub-population of sensory neurons are categorised as '*nociceptive neurons*' because they uniquely express receptors called '*nociceptors*' that are capable of detecting noxious stimuli. The process of detecting noxious stimuli is called '*nociception*'. The key point is that a '*noxious stimulus*' is fundamental to each of these characterisations. In the coming chapter I will provide reasons for doubting the metaphysical status of noxious stimuli and hence the nociceptive category as a whole.

Finally in this section, I want to mention a rarely acknowledged bias that is related to experimental design. This bias is important for my coming analysis of the nociceptive system. It is a the fact that almost all of the literature that refers to the nociceptive system *generally* talks of Aδ- and C-fibre nociceptive neurons even though these categories of neuron innervate the skin. They do not innervate the tissues of the musculoskeletal system. The nociceptive neurons which innervate muscles, tendons, ligaments, etc. are a sub-category of 'group III and IV' sensory fibres.[31] My speculative diagnosis for this terminological bias is that most pain research is conducted on the skin. The dominance of experimental designs involving the skin is hardly surprising because such experiments are easily reproduced and raise few ethical concerns in human experiments.[32] Now if this were just a labelling issue it would be of little concern but it reflects a much more worrying trend, the tendency to draw conclusions about the nociceptive system in general from experimental data that strictly speaking only licences conclusions about cutaneous noxious thermal energy. It

[31] Each of the categories of sensory neuron, Aα-, Aβ-, Aδ- and C-fibres (whether nociceptive or non-nociceptive), which innervate the skin have their musculoskeletal equivalents; group I, II, III and IV fibres, respectively.
[32] Typically, these experimental designs involve pulses of infrared heat delivered by laser (a laser evoked potential or LEP) or radiant heat delivered by a scientific instrument, called a thermode.

is likely that many scientists would reject my viewpoint, but as we shall see in the coming chapter, it is not just an unsubstantiated assertion. Despite these concerns, for the sake of clarity I will follow the prevailing trend and refer to all sensory fibres, whether cutaneous or musculo-skeletal as 'Aα-, Aβ-, Aδ- and C-fibres'.

3.4 THE CONCEPTS OF PAIN SCIENCE AND P1-P6

Much of this chapter has been devoted to clarifying three closely related conceptual models, the multidimensional model, the pain matrix and the nociceptive system. I have not yet considered whether a model based on these three conceptual models, favourably clarified, has the explanatory power to explain P1-P6. What would such a model look like?

Both the multidimensional model and pain matrix provide accounts of the system that yields *TYP*. The former presents a functional understanding of the constitution and influences on and of *TYP*. The latter frames these influences and constituents in terms of functional anatomy and it links central processing with the peripheral sensory neurology of the nociceptive system. The overriding feature of a hybrid of the multidimensional, pain matrix and nociceptive concepts is that it would look like an anatomical, physiological and functional model of the nociceptive system, its upstream connections and outputs. *TYP* is one of these outputs. Woolf and Ma capture much of this picture:

> The peripheral terminal of the mature nociceptor is its raison d'être. This is where noxious stimuli are detected and transduced into inward "generator" currents that, if sufficiently large, begin to drive action potentials along the axon to the CNS and in this way set in train the events that ultimately lead to conscious awareness of the noxious stimulus, pain (2007, p.356).

Although their emphasis is squarely on sensory input and they describe pain as the conscious awareness of the noxious stimulus, this should not be considered an unqualified endorsement of perceptualism (the position that both *Q* and *U* fulfil a perceptual function in *TYP*). The peripheral terminal of the mature nociceptive neuron[33] is its raison d'être because these neurons have *evolved* for the purpose of linking noxious stimuli with central nervous system outputs that yield appropriate responses and behaviours. *TYP* is one of these outputs. In accord with the multidimensional model, they, along with many pain scientists, would undoubtedly accept that *TYP* functions as both a conscious awareness of noxious stimuli *and* an affective mental state that motivates injury-preventing behaviour. So the hybrid would look much like mixed perceptualism/motivationalism. The difference between the two is that science is primarily concerned with the anatomy and physiology of the system(s) that yields *TYP* as an output. Unlike philosophers, scientists do not worry about accuracy conditions or about explaining the functional role of a sensory (perceptual) mental state in a gestalt-like conscious experience that motivates injury-preventing behaviour. Scientists investigate the weak correlation between *TYP* and the stimulus and they identify mechanisms that mediate this variability but they tend not

[33] Woolf and Ma's reference to the 'mature nociceptor' is an error. Nociceptors are constituents of the peripheral terminal of a *nociceptive neuron.*

to refine their conception of what it means to say that *TYP* is a conscious awareness of noxious stimuli in this light.

The missing detail about the functional relationship between awareness of noxious stimuli and motivation could be explained in a way that mirrors perceptualism; the connection between conscious awareness of the stimulus and motivation is that noxious stimuli are experienced as bad for the subject. This motivates injury-preventing behaviour. However, I do not think this accurately reflects the conceptual models of pain science.

The resources available to science have enabled scientists to identify a specific sensory system, the nociceptive system, which if not specific to *TYP* is specific to nocifense. The nociceptive system links noxious stimuli with brain areas of the pain matrix that fulfil sensory-discriminative and affective-motivational functions. These in turn are associated with the constituents of *TYP*, *Q* and *U* respectively. So there is a good reason to believe that the phenomenology of *Q* is specific to pain whatever the status of *U*. This supports Woolf and Ma's claim that *TYP* is the conscious awareness of a noxious stimulus and both perceptualism and the mixed theory. But the association of the brain areas that are involved in *U* (the ACC and IC) with affect and motivation counts against perceptualism.

I am not suggesting that science can provide all the answers to questions about *TYP* – it remains to be explained how a composite experience with perceptual and affective constituents produces injury-preventing behaviours, for example. But at the very least

science supports the position that the phenomenology of *Q* is specific to *TYP* and that the components of *TYP* fulfil perceptual and motivational functions. It is doubtful that the latter will dissuade either perceptualists or motivationalists, but it does at least strengthen the case for the mixed theory. By contrast, the former is likely to be accepted by all theorists because it provides a strong base for a fully worked out argument that the phenomenology of *Q* is specific to *TYP*. This argument would support claims that an experience of *Q* is sufficient for pain and crucially it avoids many of the constitutional/functional difficulties I set out in chapter 1, section *1.2.3*.

In this light, it is interesting that the IASP explicitly want to avoid "tying pain to the stimulus" (2014). The disconnection between *TYP* and the stimulus is manifested in the weak correlation between pain and the stimulus (P4-P6). So despite my equation of the hybrid of science with the mixed theory and Woolf and Ma's claim that *TYP* (or *Q* at least) is the conscious awareness of noxious stimuli, there is no impetus to define *TYP* in terms of this role. This is understandable because pain scientists are very concerned with the multiplicity of inputs that have the capacity to influence the transmission of nociceptive information. These modulatory factors have been the focus of much research and quite a lot of the detail of the mechanisms that mediate these factors has been worked out. But there has been little or no attempt to rationalise the claim that *Q* functions as a conscious awareness of noxious stimuli (or more weakly, that it functions as a means of consciously discriminating noxious stimuli) with the variable relationship between *TYP* and the stimulus (P4-P6). So the hybrid shares the weaknesses of the philosophical accounts I considered in chapter 2. Moreover, pain science does not address the detailed constitutional/functional issues

arising from P1-P3 so the hybrid cannot explain these issues without development. On my view, a developed version of this theory would look like a science-based version of the mixed theory.

In summary of this chapter, I have provided brief accounts of the dominant conceptual models of pain science, the multidimensional model, the pain matrix and the nociceptive system. The multidimensional model is an ambiguous mix of function and subjective nature that concerns the constitution of *TYP* as well as the influences on and of *TYP*. The pain matrix suggests a specificity that would be rejected by most (perhaps all) neuroscientists so this concept is best understood in broader terms that are consistent with its progenitor, the neuromatrix. By contrast, the nociceptive system is a peripheral sensory system, which is clearly defined in terms of the noxious stimuli it detects. Although pain science has the resources to explain many of P4-P6, with the exception of *Sprain* (P6), mechanistic explanations like modulation (some cases of P5) and maladaptive plasticity (the chronic pains of P4) are not easily accommodated into a hybrid model derived from these three models. Furthermore, the hybrid does not address several of the constitutional/functional issues raised in P2 and P3. However considered as an adjunct to the mixed theory, the multidimensional model, the pain matrix and the nociceptive system support the position that *TYP* is a composite experience constituted by phenomenal qualities with the sensory-discriminative function of representing noxious stimuli (*Q*) and an affective mental state that motivates injury-preventing behaviour (*U*). This provides a strong explanation for the many problems that fall under P2. It is less satisfactory with

respect to P3 because, like the mixed theory, it remains to be explained what role a perceptual mental state plays in the motivation of injury-preventing behaviour.

All the accounts I have considered struggle to explain P4-P6. The weakness of the correlation between *TYP* and the stimulus is a good reason to doubt that *TYP* or *Q* are perceptual in nature. In the coming chapter, I will provide a stronger reason for doubt by arguing that no sensory system has the capacity to detect noxious stimuli. This seriously undermines the mixed theory.

4 THE NOCICEPTIVE SYSTEM

The basic concept of a nociceptive system is so much a part of the fabric of pain science that it seems almost inviolate to doubt. According to this concept, the brain areas of the pain matrix including the somatosensory cortex, which is associated with the sensory-discriminative component of *TYP* (*Q*), and the anterior cingulate (ACC) and insular cortices (IC), which are associated with the affective-motivational component of *TYP* (*U*), receive input from the nociceptive system. This system is constituted by a sub-population of primary afferent Aδ- and C-fibre sensory neurons (*nociceptive neurons*) that are distinguished from non-nociceptive sensory neurons (the remaining population of Aδ- and C-fibres, as well as Aα- and Aβ-fibres) by their expression of receptors (*nociceptors*) with the unique capacity to detect noxious stimuli. So with respect to motivating injury-preventing behaviour, the crucial function of discriminating noxious intensities of energy is a peripheral affair. The corollary is that it is highly unlikely that the central nervous system (CNS) fulfils any significant role in the discrimination of such energy.[1] However, the position that the central nervous system is significantly involved in the discrimination of noxious intensities of energy has considerable power to explain problem cases (P4-P6). I argue for this position in this chapter.

[1] Although it can be argued on rational grounds that there is a significant biological advantage to over-determination, it remains "highly unlikely" that organisms would evolve both peripheral and central mechanisms for discriminating noxious stimuli.

The definitions I use in this chapter come from the IASP's Taxonomy of Pain Terms. The IASP's definitions are conspicuous in both pain science and the relevant philosophy so they reflect something of a consensus on the definitions of a 'nociceptor' and a 'nociceptive neuron'.[2] Although the arguments of this chapter amount to a criticism of these definitions, I want to be clear that my target does not concern concepts. My target is the metaphysical claim that sensory receptors can detect noxious stimuli.[3] If sensory receptors lack the ability to detect noxious stimuli then a strong case can be made for the position that CNS structures of the nocifensive functional system (*FS*) fulfil a significant role in the discrimination of noxious intensities of energy.

This chapter has four sections. Taken together, sections *4.1* and *4.2* contain an argument that has the following structure:

1) *Premise*: Nociceptors are sensory receptors with the ability to transduce and encode a noxious stimulus.
2) *Premise*: A noxious stimulus involves a relation between energy and the bodily tissues.
3) *Premise*: No sensory receptor has the ability to transduce and encode a relation between energy and the bodily tissues.
4) *Conclusion*: There are no nociceptors.

[2] For more on this see chapter 3, section *3.3*.
[3] It is fundamental to the concept of a nociceptive system.

Section *4.1* concerns the first premise. Here I define key terms including 'stimulus' and explain the interdependence of the concepts of a 'nociceptor' and a 'noxious stimulus'. The bulk of the argument in *4.2* is devoted to the key second premise. Here I conclude that a noxious stimulus is not a genuine stimulus because 'noxious stimuli' cannot be detected by sensory receptors. However, this does not imply that noxious intensities of energy are not discriminated by peripheral structures. In section *4.3*, I consider whether the information that intensities of energy are noxious is embedded in the nocifensive function of *FS* and revise the concepts of a nociceptor and a nociceptive neuron accordingly. In section *4.4*, I argue that this information cannot be embedded in *FS* because nociceptors detect energy at sub-noxious intensities and the neurons that provide sensory input to the pain matrix are not confined to high intensity stimuli because of the effects of tissue damage and adaptation to mechanical energy. The consequence of this argument is that CNS structures must be significantly involved in the discrimination of noxious intensities of energy. In the final section (*4.5*), I argue that nociceptive concepts, however they are characterised, are misleading because they suggest specificity.

4.1 'NOCICEPTORS' AND 'NOXIOUS STIMULI'

In the preceding chapters, I have written about the *detection* of stimuli or energy. In accord with the language of neuroscience, from this point on I will drop the everyday term 'detect' and substitute the technical terms 'transduce' and 'encode'. So using the jargon of neuroscience, nociceptors are specifically adapted to *transduce* and *encode* noxious stimuli (the process of encoding noxious stimuli is called '*nociception*').

Nociceptive neurons *transmit* encoded information about noxious stimuli in patterns of *action potentials* to the spinal cord, where they *synapse* with (second order) neurons which project in the spinal tracts of the medial and lateral pain systems to various areas of the brain.[4] The implication of the concept of a nociceptive system is that the discrimination of noxious stimuli is a function of specific *peripheral* anatomy and physiology.

My concern about the nociceptive system arises partly from the 'sub-category' status of nociceptive neurons, which suggests strong similarities between nociceptive and non-nociceptive Aδ- and C-fibres, and the sharp distinction that is drawn between nociceptors and non-nociceptive sensory receptors. The latter transduce and encode particular types and intensities of energy, while the former are conceived in terms of their ability to transduce and encode what I will argue is a different type of stimulus. Although it is not immediately obvious, as we shall see from this and the following section, a '*noxious* stimulus' conveys more than just a particular type and intensity of energy.

I begin by arguing that the concepts of a noxious stimulus and a nociceptor are dependent on one another; a noxious stimulus is something that is being transduced and encoded by a sensory receptor, and a sensory receptor that has the capacity to transduce and encode a noxious stimulus is a nociceptor.

A nociceptor is:

[4] Second order neurons also project to the same and other levels of the spinal cord. But this can be ignored here.

NR A high-threshold sensory receptor of the peripheral somatosensory system that is capable of transducing and encoding noxious stimuli (IASP, 2014).

And a noxious stimulus is:

NS A stimulus that is damaging or threatens damage to normal tissues (IASP 2014).[5]

Both *NR* and *NS* imply the following:

SR A noxious stimulus is the sort of thing that is being transduced and encoded by some sensory receptors.

It might immediately be objected that neither *NR* nor *NS* imply *SR*. The IASP explicitly distinguish between a noxious stimulus (*NS*) and a *nociceptive stimulus*. The latter is "An actually or potentially tissue-damaging event transduced and encoded by nociceptors" (IASP, 2014). The phrase 'An actually or potentially tissue-damaging event' appears to refer directly to a noxious stimulus because this was the definition of a noxious stimulus prior to 2008 (Loeser and Treede, 2008, p475). So the definition of a 'nociceptive stimulus' translates to '*a noxious stimulus that is being*

[5] Other definitions are possible, but all reflect the widely held view that noxious stimuli are stimuli that have the capacity to damage bodily tissues. The IASP have reformulated their definition of a 'noxious stimulus' four times, but these revisions have not been substantive. In their first taxonomy of pain terms, they defined it as "a tissue damaging stimulus" (Bonica, 1979, p.251). This was superseded by a "stimulus that is damaging to normal tissues" (IASP, 1986) and recently the IASP's Task Force on Taxonomy suggested an "actually or potentially tissue-damaging event" (Loeser and Treede, 2008, p.475).

transduced by a nociceptor'. This is in accord with Loeser and Treede's statement that nociceptive stimuli are a "subset of noxious stimuli" (2008, p475).[6] As nociceptors are the sensory receptors that uniquely transduce and encode noxious stimuli and some noxious stimuli are not transduced and encoded by any sensory receptors, neither *NR* nor *NS* implies *SR*.

By this objection the word 'stimulus' is being interpreted in the broad biological sense that it is something that is producing a response in living tissues; e.g. ultra-violet light (UV) that is producing a response (melanin production, say) in human tissues is a stimulus in this broad biological sense. So if UV is damaging tissue and producing a physiological response it is a noxious stimulus that does not fall under *SR* because it is not being transduced and encoded by (human) sensory receptors.

There are several anomalies in the IASP's definitions of nociceptive terms so it is unclear whether the IASP intend this broad understanding of a 'stimulus', despite the distinction they make between a nociceptive and a noxious stimulus. Here is an example: In the notes that accompany the recommended revisions discussed at the 2007 meeting of the Task Force on Taxonomy, Loeser and Treede write of a noxious stimulus that, "there are some types of tissue damage that are not detected by any sensory receptors" (2008, p475). This is a surprising claim, as sensory receptors do not detect tissue damage.[7] In view of the weight of the evidence, I take Loeser and

[6] This quotation comes from the report of a meeting of the IASP's Task Force on Taxonomy in 2007, at which several revisions were recommended.

[7] Indeed, Loeser has written in contradiction that nociception "is defined as the activation of A delta and C fiber axons by *mechanical, thermal and chemical energies* which are capable of damaging body tissues" (1991, p215; my emphasis). It is unlikely that Loeser has changed his mind because the

Treede's quotation to be loose talk, which should be interpreted as: 'some noxious stimuli are not detected by any sensory receptors in the sense that energy is noxious because it is damaging the tissues and it is producing a physiological response (other than stimulating a sensory receptor)'.[8] It is certainly correct to think of a stimulus in the broad sense that energy at a bodily location has the potential to produce many different physiological responses. But the key point is that we are concerned with a *particular* response, transduction and encoding by a sensory receptor because our focus is on the relationship between the nociceptive system and *TYP*. Consequently, non-nociceptive responses to energy are irrelevant, the only *relevant* understanding of a 'stimulus' is narrow:

S *E* is a stimulus iff *E* is being transduced and encoded by a sensory receptor.[9]

It may be that the IASP are intending 'a stimulus' to be interpreted in a broad biological sense rather than as stated in *S*, but my hunch is that their distinction between a nociceptive and a noxious stimulus reflects the widespread conflation of a 'stimulus' and a 'potential stimulus'. Thermal energy at 50^{o}C at a bodily location is

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evidence remains that nociceptors transduce energy and not damage. (See chapter 2, section *2.1.2* for my argument that tissue damage is not detected by sensory receptors.)

[8] It is important to note that tissue damage is an *effect* of thermal energy at 50^{o}C at a bodily location (say). It is not a response to that energy. Hence, the energy is the *cause* of the damage, it is a mistake to describe the energy as a stimulus with respect to damage. A biological response is an active process. The damaging effect of energy is the breakdown of tissue. Note that even though damage is not strictly speaking a response it can be a stimulus. For example, tissue damage produces responses like phagocytosis and damaged neurons can depolarise (transmit action potentials) spontaneously.

[9] There is a complex relationship and a distinction to be drawn between the transduction and encoding of a stimulus into action potentials and the binding of chemicals to molecules (gates, pores, channels, etc.) that results in an increase or a decrease in the sensitivity of the neuron. I am *not* using the term 'stimulus' to refer to the chemicals that are binding to these sensitising molecules. My reason for this is that the stimuli that are being transduced and encoded lead directly to the transmission of action potentials to the central nervous system. Changes in sensitivity reflect modulation by diverse influences, but they do not *directly* provide input to the CNS. (Modulation is a problem for perceptualism and mixed perceptualism/motivationalism, see chapter 2, sections *2.1.5* and *2.3*; and for more on modulation see Tracey and Mantyh, 2007.)

noxious because it is damaging tissue, but by *S* it is only a stimulus if it is being transduced by a sensory receptor. If it is not being transduced and encoded by a sensory receptor it is not a noxious stimulus but it is a potential noxious stimulus because it could be transduced and encoded by a sensory receptor (by *NR*). The use of the present continuous tense in *S* distinguishes a stimulus from a potential stimulus. Thermal energy at 25°C is a stimulus just in case it *is being* transduced and encoded by a sensory receptor, while thermal energy at 25°C is a *potential stimulus* if it merely *could be* transduced and encoded. By contrast, something that could not be transduced and encoded by any of an organism's sensory receptors is not a potential stimulus (in the relevant sense) for that organism and of course it is not a stimulus. In more detail:

PS *E* is a potential stimulus for an organism iff *E* it merely could be transduced and encoded by at least one of that organism's sensory receptors.

OS *G* is not a potential stimulus (*S*) for an organism because it cannot be transduced and encoded by at least one of that organism's sensory receptors.

Even though UV that is damaging tissue is noxious by definition, it is not a stimulus (it falls under *OS*) because it cannot be transduced and encoded by human sensory receptors. Therefore, it is not a counterexample to my claim that both *NR* and *NS* imply *SR*.

As both *NR* and *NS* are committed to *SR*, 'transduction' is another crucial component of this conceptual scheme.[10] Transduction is:

Trans The neurophysiological process by which a sensory receptor converts a type of energy into the units of energy (electro-chemical action potentials) that are utilised by the nervous system.

The mutual dependence of a 'noxious stimulus' and a 'nociceptor' is now clear. A member of a sub-class of *E* is a noxious stimulus if and only if it is being transduced and encoded by (i.e. iff it is stimulating in the relevant sense) a sensory receptor. A sensory receptor with the ability to transduce and encode a noxious stimulus is a nociceptor. The obvious distinction between the members of this sub-class and the remaining population of *E* is that the former are noxious stimuli while the latter are not noxious stimuli (I will refer to these as 'innocuous stimuli').

4.2 WHAT IS A 'NOXIOUS STIMULUS'?

Even though the distinction between noxious and innocuous stimuli appears unambiguous, *NS* is open to at least three different interpretations. As each interpretation must involve a member of the sub-class of *E* being transduced and encoded by a sensory receptor, necessarily each interpretation of a noxious stimulus involves what I call a '*stimulus event*', i.e. transduction and encoding by a sensory

[10] Note that by *SR*, encoding is a constituent of both *NR* and *NS*. Encoding is the neurological process of organising action potentials into a pattern or code that represents something about the energy that has been transduced. The inclusion of encoding in this analysis would only add an unnecessary layer of complication so it has not been included.

receptor. By the first interpretation, a noxious stimulus is a stimulus event constituted by the transduction and encoding of the relation between a particular type and intensity of energy at a bodily location and the noxious threshold of the tissue at that location. By the second interpretation, a noxious stimulus is a stimulus event constituted by the transduction and encoding of the tissue-damaging disposition of a type and intensity of energy at a bodily location. The final interpretation of a noxious stimulus involves two distinct events: the transduction and encoding of a particular type and intensity of energy by a sensory receptor (this is the '*stimulus event*'), and the more or less simultaneous damaging of tissue by that energy at or very close to the location of the stimulus event (the '*noxious event*'). This section is divided into three sub-sections. In the first sub-section, I present each of these options. In the second sub-section, I reject the first two interpretations of *NS*, and in the third sub-section, I reject the third interpretation.

4.2.1 Three interpretations of a 'noxious stimulus'

By the first understanding, a noxious stimulus is constituted by a single event:

E1 A noxious stimulus is a stimulus event, where the stimulus event is constituted by a relation between a particular type and intensity of energy at a bodily location and the noxious threshold of tissue at that location.

This understanding places a considerable demand on our current understanding of sensory physiology. By *S*, a noxious stimulus must be something that is being

transduced and encoded by a sensory receptor, and by *Trans* a stimulus event is a relation between particular properties of energy and a sensory receptor that is adapted to transduce in response to these properties. So it is clear that on a conventional understanding of transduction sensory receptors do not transduce the further relation between the energy that has been transduced and the noxious threshold of the tissues. Therefore, *E1* requires the revision of *Trans*.

On the second reading, a noxious stimulus is constituted by a single event:

E2 A noxious stimulus is a stimulus event, where the stimulus event is constituted by a relation between the transduction capacities of a sensory receptor and the noxious disposition of energy.

This reading differs subtly from *E1* because the stimulus event is conceived as a relation between a specifically adapted receptor and an intrinsic property of energy at a bodily location, the noxious disposition of a particular type and intensity of energy at that location. If this disposition is a proper intrinsic property of energy, *E2* does not obviously require a revisionary approach to *Trans*.

The final understanding of *NS* is that a particular type and intensity of energy at a bodily location is both a stimulus and it is damaging or threatening damage to the tissues at that location. So a noxious stimulus is constituted by distinct noxious and stimulus events:

E3 A noxious stimulus is constituted by noxious and stimulus events, where the noxious event is the damaging of, or the threat of damage to, a tissue by energy and the stimulus event is the more or less simultaneous transduction and encoding of the particular type and intensity of the energy that is involved in the noxious event.

On this understanding, energy at a bodily location is a constituent of distinct but more or less *simultaneous* relations with the body. The noxious event is a relation between a particular type and intensity of energy at a bodily location and a threshold that marks the division between innocuous and threatening intensities of energy (the '*noxious threshold*'[11]) at that location. The stimulus event is a relation between the energy at (more or less) the same bodily location where the noxious event is occurring and the specific transduction and encoding capacities of a sensory receptor at that location. So a type and intensity of energy is a noxious stimulus just in case it *is* damaging the tissues, and it *is* being transduced and encoded by a sensory receptor (i.e. just in case the energy is a constituent of both noxious and stimulus events). To illustrate, consider thermal energy at 25^{o}C, ultraviolet light and thermal energy at 50^{o}C. On the one hand, thermal energy at 25^{o}C that is being transduced and encoded by a sensory receptor is a stimulus event, but as the energy is below the noxious threshold it is not involved in a noxious event so it is not a noxious stimulus. On the other hand, UV light that is damaging the tissues is a noxious event, but the energy is not involved in a stimulus event because it cannot be transduced and encoded by human sensory receptors. So it is not a noxious stimulus. By contrast, thermal energy at 50^{o}C at a

[11] I define the noxious threshold in these terms in chapter 2, section, *2.1.2* and discuss problems associated with this concept in chapter 6, section *6.2.1*.

bodily location is clearly a noxious event,[12] and the energy is a potential stimulus (because it could be transduced and encoded by at least one of the subject's sensory receptors). Therefore, if there is an appropriately adapted sensory receptor at (or very close to) the location of the noxious event then it is highly likely the energy is also involved in a stimulus event. In which case, the energy would be a noxious stimulus.

Each of *E1*, *E2* and *E3* provides a means of distinguishing the members of the sub-class of stimuli (the sub-class of *E*) that are noxious stimuli from other innocuous stimuli. Unfortunately, each also has significant problems.

4.2.2 Problems with E1 and E2

As I have mentioned above, the understanding of a 'noxious stimulus' expressed by *E1* is inconsistent with *Trans*. If transduction were the conversion of energy and only energy then the relation between a particular intensity of a particular type of energy and the damage threshold of a tissue would have to be energy. I have no idea how to make sense of the claim that this relation could be energy, so either *E1* is fatally inconsistent with *Trans* or transduction is also the process of converting a relation between a particular intensity of a particular type of energy and the noxious threshold of a tissue into action potentials. As there is no evidence that sensory receptors have this latter capacity a noxious stimulus is not the relation expressed in *E1*.

[12] By comparison with the noxious threshold, the 'damage threshold' is easily characterised. Bodily tissue at a given location begins to damage at this threshold. For thermal energy, it is approximately 47^{o}C throughout the body. See chapter 2, fn.10.

The idea that a noxious stimulus is the noxious disposition of some intensities of some types of energy would appear to be consistent with *Trans* if such a disposition were an intrinsic property of energy. If it is not, then *E2* collapses into *E1*. Thermal energy at 50°C can only be said to have a noxious disposition *because* it so happens that the integrity of tissue is threatened by thermal energy at or above 42°C (say). In other words, the claim that some intensities of some types of energy have a noxious disposition depends on a relation between the tissues and energy. If the noxious threshold of tissue were 52°C, thermal energy at 50°C would retain *all* its intrinsic properties but lack the noxious disposition.[13]

Some will not be convinced by this argument.[14] Dispositional properties are controversial precisely because arguments like this cannot settle the matter. Even though my view is that dispositional properties are metaphysically dubious, I want to emphasise that dispositions are not my target here. So *if* energy has a noxious disposition, the matter at hand is whether this disposition can be transduced and encoded by sensory receptors. So with the possibility that energy has a noxious disposition in mind, there are two problems. First, it would require a further argument to persuade us that a noxious disposition is the sort of objective property that can be transduced by a sensory receptor. According to current science sensory receptors

[13] 'All' is intended to convey the thought that in a close possible world, where the only distinguishing feature is noxious thresholds of 42°C and 52°C, there would be no difference in the *intrinsic* properties of thermal energy.

[14] Michael Tye and Manolo Martinez number amongst those who may not be convinced. Tye writes, "aptness to harm, is certainly an objective property" (2005b, p.167). 'Aptness to harm' sounds like a noxious disposition, but Tye is not entirely clear of what it is a property. At first, he seems to be referring to an objective property of tissue damage, but then he writes of the prostaglandins released by damaged tissue causing a shift in the "body landscape" that is "not good for the subject" (2005b, pp.167-168). Given the ambiguity, it should be added that he might not endorse the claim that a noxious disposition is an *intrinsic* property of energy. Manolo Martinez explicitly supports Tye's position (2011, p.72).

transduce energy of particular types at particular intensities so I doubt the availability of an empirically sound argument to this effect.

Second, *E2* amounts to the claim that a noxious disposition distinguishes types and intensities of energy that could be transduced and encoded by nociceptors from intensities and types of energy that could not be transduced and encoded by nociceptors because they lack this disposition. In other words, nociceptors are specifically adapted to transduce and encode the noxious disposition of energy. Non-nociceptive receptors lack this specific adaptation. The only way that this claim can work is if there is a fixed noxious threshold. By this I mean the relationship between energy and the noxious threshold of tissues is fixed for all tissues, from subject to subject and from time to time.[15] For example, the noxious threshold for thermal energy is always 42^{o}C because tissue is threatened by temperatures that are close to the damage threshold, which is always 47^{o}C. Therefore, thermal energy at 50^{o}C has a noxious disposition, while thermal energy at 25^{o}C lacks this disposition. Note that for thermal energy possession of a noxious disposition is consistent with *PS*. Thermal energy at 50^{o}C is a potential noxious stimulus *because* it has a noxious disposition.

The problem is that there is not a fixed noxious threshold for mechanical energy because many species have the ability to adapt to mechanical energy. This ability is exemplified by tissue responses to increases and decreases in exercise. For example, the noxious threshold for muscle fibres increases in response to specific training.[16] Hence, an intensity of mechanical energy that had the potential to damage a specific

[15] This is a very important theme for my thesis. I explore a different aspect of the question of a fixed relationship between the tissues and energy in coming section.
[16] I provide more detail about these effects in the following section.

tissue at one time may not have the potential to damage that tissue at another time, and *vice versa*. Furthermore, the noxious threshold for mechanical energy varies from tissue to tissue for a given subject and from subject to subject for a given tissue. The consequence being that many (perhaps most) intensities of mechanical energy have the potential to threaten damage to a tissue of a subject. If an intensity of mechanical energy could threaten damage to a tissue, then that intensity of energy has a noxious disposition. Hence, many (perhaps most) intensities of mechanical energy have a noxious disposition. Now we are always absorbing and generating mechanical energy and as much of this energy has a noxious disposition, nociceptors throughout the body will be transducing and encoding this disposition. Even if we take account of the weakness of the correlation between nociceptive activity and *TYP*[17], this understanding of *E2* predicts that most subjects would be experiencing *TYP* from multiple bodily locations most of the time. This prediction is clearly contradicted by subjective experience.

The response of relating the noxious disposition of mechanical energy not only to the subject, but also to particular tissues at particular times is not available because of the physiological requirements. Transduction is a physiological response to intrinsic properties of energy. If a given intensity of mechanical energy's possession or lack of a noxious disposition depends on the noxious thresholds of different tissues of a given subject at a given time, then a noxious disposition is a relation between the energy and the subject. So this response suffers the same problem as *E1*, it is inconsistent with *Trans*.

[17] The weakness of this correlation gives problems P4-P6; see chapter 1, section *1.2.5*.

4.2.3 The problem with E3

E3 has a distinct advantage over *E1* and *E2*, it is clearly consistent with *Trans*. Transduction is a response to a particular type of energy at particular intensities. Thermal energy at 50^{o}C, which is potentially noxious because it exceeds the noxious threshold, is a potential stimulus because it can be transduced and encoded by a sensory receptor. As such *E3* makes no demands on the transduction process over and above the demands posed by uncontroversial stimuli like thermal energy at 25^{o}C. It also provides a principle for distinguishing noxious from non-noxious stimuli; noxious stimuli are constituted by *both* stimulus and noxious events, while innocuous stimuli are constituted by just a stimulus event. This straightforward distinction is appealing but it is also a glaring disadvantage. Under *E3*, a noxious stimulus is not a genuine stimulus because the noxious event (the crucial relation between energy and the damage threshold of tissue) is independent of the transduction process. For this reason *E3* fails to distinguish nociceptors from non-nociceptive sensory receptors. To see this consider that *all* classes of sensory receptor transduce and encode particular types and intensities of energy. There is a difference between sensory receptors with the capacity to transduce and encode thermal energy above 50^{o}C (50^{o}C>), 35^{o}C>, and 20^{o}C>. The '*thermal thresholds*' of these receptors mark differences in their respective capacities to transduce thermal energy and so they provide a principle for distinguishing between sensory receptors. Under a classification like this a receptor with a thermal threshold of 50^{o}C would be distinct from a receptor with a threshold of 35^{o}C for the same reason that the latter is distinct from a receptor with a threshold of

20°C. There is nothing in this to distinguish the nociceptor (the receptor with the threshold of 50°C) from the other two types of (non-nociceptive) receptors.

The definition of a nociceptor as a 'high-threshold sensory receptor' (see *NR*) does not solve this problem.[18] A receptor with the capacity to transduce and encode thermal energy at 50°C> has a higher threshold than a receptor with a threshold of 35°C, but having a high threshold in relation to other receptors cannot distinguish nociceptors from other classes of sensory receptor in the relevant way. The relevant difference between thermal energy at 50°C and 35°C is that the former is while the latter is not high in relation to the damage threshold (for thermal energy) of tissue, not that thermal energy at 50°C is high in relation to thermal energy at 35°C.[19] Even if the reference to 'high' is dropped in favour specifying specific thresholds (like a thermal threshold of 42°C) the sensory receptor is not discriminating that thermal energy at 42°C is noxious. As the relation between energy and the tissues cannot be transduced by any sensory receptors noxious stimuli are not genuine stimuli (they fall under *OS*). Consequently, the ability to transduce and encode noxious stimuli or high intensity stimuli cannot be used as a means of distinguishing nociceptors from non-nociceptive sensory receptors but more importantly, sensory receptors cannot transduce and encode noxious intensities of energy.

[18] The term 'high threshold' is misleading because it is too exclusive. Low intensities of thermal energy (i.e. cold) are also constituents of noxious events. So some nociceptors are low threshold receptors if 'high-threshold' is taken to refer to relative intensities of energy. Understanding 'high threshold' in terms of exceeding the damage threshold is less problematic.

[19] To be clear on this, by 'relevant' I mean that the *relevant question* is whether sensory receptors can *transduce* that an intensity of energy is high in relation to the noxious threshold, not that it is high in relation to other intensities of energy.

4.3 THE NOCICEPTIVE SYSTEM – A REVISION

The argument that no sensory system is specifically adapted to transduce and encode noxious *stimuli* does not amount to an argument that no peripheral system is specifically adapted to *discriminate* noxious intensities of particular types of energy. The formulation in *NR* follows the standard approach, which is to classify sensory receptors in terms of the stimuli they are adapted to transduce (their 'adequate stimuli'). As a noxious stimulus is not the sort of thing that can be transduced by a sensory receptor there are no nociceptors, and without nociceptors there is no nociceptive system as conceived by the IASP.[20]

The problem is that the noxious and stimulus effects of an intensity of energy at a bodily location are independent of one another in the sense that the noxious event does not involve the stimulation of a receptor and the stimulus event does not involve the damaging of tissue. However, this does not mean there is no connection between the former and the latter. If we have a functional system that receives sensory input about particular types and intensities of energy and it yields outputs that function as protective responses to the energy, then we have evolved the ability to at least utilise the ability to transduce and encode particular types and intensities of energy *because* they are at or exceed the noxious threshold.[21] Here 'because' implies the evolutionary advantage of having receptors that are specifically adapted to transduce and encode

[20] Because all the nociceptive terms in the IASP's taxonomy ('nociceptor', 'nociceptive neuron', 'nociception', 'nociceptive stimulus' and 'nociceptive pain') are defined in terms of a noxious stimulus.

[21] I say 'we have evolved the ability to at least utilise the ability to transduce…' rather than 'we have evolved the ability to transduce…' because there are reasons to think that the ability to discriminate energy at or above the noxious threshold *may* have evolved for a purpose other than nocifense. (See chapter 5, section *5.1* for more on this.)

energy that is close to or above the damage thresholds of tissue. There is no doubt we have such a functional system (this is '*FS*' – the 'nocifensive functional system').[22] So we can say that nociceptors function as a means of discriminating noxious intensities of particular types of energy.[23]

The insight provided by consideration of adaptation concerns the discriminatory function of a specific class of sensory receptor in a functional system that has the capacity to yield advantageous outputs. In this case, a sub-class of thermo- and mechanoreceptors fulfils a discriminatory role in a functional system (*FS*) with the capacity to yield injury-preventing responses and behaviours in response to intensities of thermal and mechanical energy that are damaging or threatening damage to the tissues. So the transduction capacities of the sensory receptors expressed by the neurons that provide input to *FS* must be determined by the nocifensive function of the system. In more detail, a nociceptor is a sensory receptor that is a constituent of *FS* because it has a particular structure which determines its ability to transduce energy at or above the noxious threshold, where 'because' implies the evolutionary advantage of linking stimuli at these intensities with nocifensive responses and behaviours. This suggests a different understanding of a nociceptor:

NR′ A sensory receptor of the peripheral somatosensory system that is specifically adapted to be a constituent of a system that yields nocifensive outputs (*FS*)

[22] See chapter 2 section *2.1.6*, and chapter 5 section *5.6*.
[23] This strategy is suggested by Sir Charles Sherrington's explicit reference to adaptation in his original (1906) conception of a nociceptor. On his view, nociceptors are sensory receptors which are specifically adapted to detect stimuli of a sufficient intensity to threaten the integrity of bodily tissues (Snider and McMahon, 1998).

because the receptor has the capacity to transduce and encode intensities of particular types of energy that are at or above the noxious threshold.

By *NR′*, a nociceptor is like any other type of sensory receptor, all sensory receptors transduce and encode particular types and intensities of energy. But, unlike non-nociceptive sensory receptors, nociceptors are constituents of *FS* because they have the ability to transduce intensities of particular types of energy that are relevant to nocifense.[24] I close this section by briefly discussing the impact this analysis has on the concept of a 'nociceptive neuron', which features prominently in the next section of this chapter.

The IASP's definition of a nociceptive neuron as a "central or peripheral neuron of the somatosensory nervous system that is capable of encoding noxious stimuli" (2014) is problematic because noxious *stimuli* cannot be encoded; there are no noxious stimuli in the relevant sense of *S*.[25] By *NR′*, intensities of energy (like thermal energy at 50^{o}C) are discriminated and encoded by nociceptors. However, this does not imply that the additional information that *energy* is noxious cannot be encoded by peripheral neurons. That is by patterns of action potentials that arise in the neuron from the temporal and spatial summation of activity in the receptors expressed by that neuron.

There is evidence that some quite complex features of mechanical stimuli such as the curvature and texture of surfaces as well as the direction of a stroke are encoded into the timing of action potential spikes rather than their rate (Saal et al, 2009, Johansson

[24] This does not mean that the receptors that transduce energy at or above the noxious threshold are constituents of *FS* and no other functional system. (See chapter 5, section *5.6*.)
[25] See page 139.

and Flanagan, 2009). By comparison with these examples, the encoding of noxious intensities of energy seems to be a simple task. However, Johansson and Flanagan are claiming that it is the relative timing of spikes in "ensembles of tactile afferents" (2009, p351) that bears coded information of this sort. This is a telling point. Johansson and Flanagan's 'codes' do not result from a process of encoding by a sensory receptor, nor are they something that takes place in a primary afferent neuron. There are four peripheral elements to this 'encoding': stimulus intensity, stimulus type, stimulus location and stimulus timing. The evidence is that sensory neurons play a significant part in encoding stimulus intensity, and perhaps they are also involved in encoding stimulus type and location,[26] but timing is the crucial element in the representation of curvature, direction of stroke and so on. Johansson and Flanagan are describing a mechanism by which the temporal interval between the receipt of inputs from the fibres that constitute these ensembles of tactile afferents is interpreted as the curve of an object or the direction of stroke. Importantly, this is a discriminative function of the CNS.

[26] It is questionable whether complex patterns of action potentials that code energy type, intensity, location, and noxiousness *could* be maintained across synapses. Synapses are bridged by the release of a neurotransmitter substance (glutamate in the majority of cases) in response to action potentials in the pre-synaptic neuron. This substance binds to receptors (in the case of glutamate, the AMPA and kainate sub-types of the ionotropic glutamate receptor) at the distal terminus of the post-synaptic neuron. If sufficient quantities of glutamate bind to post-synaptic receptors then action potentials are generated in the post-synaptic neuron. I doubt a mechanism like this could maintain complex patterns of action potentials. Neuroanatomical specificity is a better explanation than encoding for the ability to experience thermal and mechanical *TYP*s and for felt location because it does without this complexity. It is consistent with the evidence that the body is spatially represented in the somatosensory cortex so that the location of a stimulus (say) is preserved by anatomical specificity. Action potentials that originate in a first order sensory neuron are transmitted to the relevant brain area that represents the bodily location of the distal terminus of that peripheral neuron. Although nothing really hangs on this matter (hence I have consigned it to a footnote), I have laboured the point because it is a further reason to doubt the extent of the discriminatory function of first-order neurons. For more on somatosensory representations, see Flor (2002), and for the related concept of 'body schema', see Gallagher (1986).

Likewise, if intensities of energy are represented by the timing of impulses in a single neuron and/or the summation of impulses across ensembles of neurons, CNS structures of *FS* are required to discriminate which patterns are of relevance for nocifense; i.e. peripheral neurons are not encoding that energy is noxious. This reinforces my position that the CNS fulfils a significant role in the discrimination of noxious intensities of energy.

It is more promising to base the definition of a nociceptive neuron on the nocifensive function of *FS*. Something of the following sort comes to mind: 'A central or peripheral neuron of the somatosensory nervous system that is a constituent of *FS* because it is specifically adapted to transmit encoded information about intensities of thermal and mechanical energy that are at or above the noxious threshold'. This definition follows the IASP's lead by treating central and peripheral neurons in exactly the same way. In my view, this is a mistake. Higher order neurons may transmit or even encode the information that energy is noxious but primary afferent (first-order) neurons, unlike higher order neurons, express sensory receptors. It is at least explanatorily helpful to preserve this distinction in the definition of a nociceptive neuron.[27] As the link between the neuron and nocifensive function can be preserved by defining a nociceptive neuron in terms of *NR′* and this strategy enables a distinction to be made between peripheral and central neurons, the following is a more apposite definition of a 'nociceptive neuron':

[27] It is important to add that this suggested definition does not imply that peripheral or central pathways are specific either to *FS* or to sensory receptors that have stimulus thresholds at or above the noxious threshold. The account of *FS* I provide in chapter 5, section *5.6* embraces these possibilities.

NN A peripheral neuron of the somatosensory nervous system that expresses nociceptors.

That *NN* relies on the coherence of the category 'nociceptor' is as it should be. Without receptors with an ability to transduce in response to the intensities of thermal and mechanical energy that are at or above the noxious threshold and *FS*, there would not be nociceptive neurons.

It is consistent with *NR'*, *NN* and *FS* to claim that as intensities of energy, like thermal energy at 50^{o}C, are discriminated peripherally and utilised in a functional system that is specific to nocifensive responses and behaviours (*FS*), the information that energy is noxious is *embedded* in the function of *FS*. In other words, within *FS* the peripherally encoded information that there is thermal energy at 50^{o}C in a bodily location *is as good as* the receipt (by the CNS) of the information that the energy at this location is noxious to a particular degree. Consequently, the CNS fulfils no significant active role in the discrimination of noxious intensities of energy.

4.4 THE DISCRIMINATION OF NOXIOUS INTENSITIES OF ENERGY

In sections *4.2* and *4.3*, I have argued that noxious stimuli cannot be transduced and encoded by sensory receptors and that the information that energy is noxious is not encoded by peripheral neurons, respectively. In this section, I will argue that the information that energy is noxious is not embedded in the nocifensive function of *FS*.

My argument is in two parts. The first part (in *4.4.1*) concerns evidence that the sensory receptors which are constituents of *FS* transduce and encode in response to intensities of energy that are below the noxious threshold and the second concerns the variability of the noxious threshold. This second part is focused on changes in the noxious threshold due to both tissue damage (in *4.4.2*) and tissue adaptation (in *4.4.3*).

4.4.1 The nature of the sensory input to FS

In an influential paper Bud Craig writes, "the empirical mechanical, thermal and polymodal thresholds of small-diameter afferents extend broadly across the 'pain' threshold in all tissues" (Craig, 2002, p.657). By this Craig means that some nociceptive neurons encode and transmit action potentials in response to intensities of energy that are below the noxious threshold.[28] The implication is that either nociceptive neurons express non-nociceptive sensory receptors or nociceptors transduce in response to energy that is below the noxious threshold or both. Either way, the evidence is that input about innocuous intensities of energy is transmitted to *FS*.

Given this evidence, if the information that an intensity of energy is at or above the noxious threshold is embedded in the nocifensive function of *FS*, then *ceteris paribus* all the input to *FS* would trigger nocifensive responses whether or not the intensity of

[28] Although Craig's reference to 'small-diameter afferents' seems to suggest Aδ- and C-fibres more generally, his talk of the 'pain threshold' narrows his target down to the sub-class of Aδ- and C-fibres that is categorised as nociceptive neurons.

the stimulus was at or above the noxious threshold.[29] The implication is that *FS* would very frequently be triggering nocifensive responses to innocuous intensities of energy. As one of these responses would be *TYP*, we would very frequently be experiencing pain in response to innocuous intensities of energy. This is not the case so the claim that the information that intensities of energy are noxious is embedded in the nocifensive function of *FS* is false.

To add weight to the evidence for this argument, there is also good reason to think that non-nociceptive Aδ- and C-neurons and perhaps Aβ-neurons are anatomically connected with *FS* because first order nociceptive and non-nociceptive neurons converge on second-order 'wide dynamic range' (WDR) neurons at the level of the first synapse. WDR neurons project to areas of the pain matrix.[30] If this is right, then *FS* receives input about a wide range of intensities of energy. In the following two sub-sections it will become clear that information about a wide range of intensities of energy is of potential relevance to *FS* because noxious thresholds rise and fall as a consequence of tissue damage and adaptation. The evidence is that the receptors that are classed as 'nociceptors' transduce and encode intensities of energy that are below the noxious threshold and that *FS* receives input from 'non-nociceptive' neurons. All of this suggests that *FS* monitors input and discriminates which is noxious.

4.4.2 Changes in the damage threshold due to tissue damage

[29] The *ceteris paribus* clause is an acknowledgement that modulatory factors have an impact on whether or not *FS* triggers nocifensive responses.
[30] Remember too that the brain areas that constitute the pain matrix are not specifically for pain. (See chapter 3, section *3.2*.)

The IASP define a noxious stimulus as a "stimulus that is damaging or threatens damage to normal tissues" (IASP 2014).[31] I take it that the reference to 'normal tissue' is equivalent to 'undamaged tissue'. In which case, a stimulus that is further damaging or threatening further damage to tissue that is already damaged is not, by this definition, a noxious stimulus. Given that the nociceptive terms in the IASP's taxonomy are all defined in terms of a noxious stimulus, the implication of this is that the nociceptive system has not been ascribed a role in the discrimination of intensities of energy that are having a noxious effect on damaged tissue. This is a strange omission.

The term 'noxious' connotes harm. If an intensity of energy at a bodily location where there is damaged tissue were having a *harmful* effect, the effect would be noxious. So tissue has a damage threshold (and a noxious threshold) whether it is damaged or not. Additionally, from a functional perspective it makes sense that *FS* is involved in responses and behaviours that facilitate recovery when tissue is damaged. The evidence provided by those unable to experience *TYP* is that *TYP* performs an essential function in preventing further damage. [32] So *FS* is involved in motivating injury-preventing behaviours that enables tissue damage to heal. As the function of nociceptive neurons is to encode noxious stimuli and these are not noxious stimuli, it is an open question whether, according to the IASP, energy at intensities that is noxious for damaged tissue is transmitted to *FS* by nociceptive or non-nociceptive neurons.

[31] See section *4.1*, this chapter.
[32] This is P3, chapter 1, section *1.2.4*.

Consideration of the temporal aspect of noxious events is also problematic for the IASP's narrow conception of a noxious stimulus. When someone is pricked by a needle the intensity of the mechanical energy being imposed by the needle reaches the noxious threshold at time t_1. At t_2, it reaches the damage threshold. By t_3, the needle has penetrated to a particular depth so damage is present from t_2 to t_3. Between t_1 and t_2, a noxious stimulus is present and the intensities of energy are transduced and communicated to *FS* by nociceptive neurons, but from t_2 to t_3, as the tissue becomes further damaged, the stimulus is not noxious and the role of nociceptive neurons is unclear.

I am sure that the IASP would not consider the latter to be a case of a noxious intensity of energy being imposed on *damaged* tissue, and it would be reasonable for them to dismiss this as an example of excessive analysis. I presume the issue as they see it concerns the release of so-called algogenic substances (like prostaglandins,[33] nerve growth factor, histamine, etc.) by damaged tissue. These substances sensitise neurons so they transmit intense barrages of action potentials in response to relatively low intensity stimuli. Although, algogenic substances have profound effects on the transduction capacities of sensory receptors and the thresholds at which action potentials are generated, it takes a little more time than the time from t_2 to t_3. Nevertheless, even with the caveat 'once these effects have begun to occur', it remains the case that by the IASP's nociceptive terms the function of nociceptive neurons in the discrimination of intensities of energy that are harming or threatening to harm damaged tissue is ambiguous. However, this conceptual matter is not the

[33] Michael Tye refers to these in his 2005b. See fn.14 this chapter.

issue. The point is that *FS* is effective in motivating behaviour that avoids or minimises *further* damage. In the presence of tissue damage, intensities of energy that would not have been sufficient to threaten tissues become a threat. In other words, these noxious intensities are 'low' in relation to the intensities of energy that are noxious for undamaged tissue. The change in stimulus thresholds and the proclivity to fire off higher frequencies of action potentials to *FS* is an adaptive response of neurons to a reduction in the damage threshold in the presence of tissue damage whatever the classification of those neurons.

By *NR'*, the sensory receptors involved in detecting this energy are nociceptors because any receptor that has the capacity to transduce and encode energy at or above the noxious threshold and is a constituent of *FS*[34] is a nociceptor and by *NN*, the neurons that express these receptors are nociceptive. But this also highlights the broad scope of the categories 'nociceptor' and 'nociceptive neuron' by *NR'* and *NN*. It includes receptors that have stimulus thresholds well below the noxious threshold of undamaged tissue and neurons that transmit information about intensities of energy that are well below the noxious threshold in undamaged tissue. This also strongly suggests that CNS structures of *FS* fulfil a significant function in discriminating noxious intensities of energy.

[34] I am ignoring the complication posed by allodynia here. *Allodynia* is an experience of *TYP* in response to the stimulation of non-nociceptive receptors in undamaged tissue (Loeser and Treede, 2008, p.476). Although allodynia refers to neurons with sensory endings in undamaged tissue these nerve endings are *close to* areas of damaged tissue. Allodynia is explained as a response to tissue damage in which there is an anatomical change at the level of the first synapse so that Aβ-fibres have access to the tracts of the medial and lateral pain systems. So current opinion is that novel connections between Aβ- (classified as 'non-nociceptive') neurons are formed during tissue damage.

4.4.3 Changes in the damage threshold due to adaptation

Although, I have used thermal energy as a means of illustrating discussion and analysis throughout my thesis, my conclusion that noxious intensities of energy are discriminated by CNS structures of *FS* applies equally to mechanical energy. Thermal energy has been a very useful explanatory tool because the damage threshold for thermal energy in undamaged tissue is fixed (at 47°C). It is fixed because we cannot *adapt* to thermal energy.[35] By contrast, the damage threshold for mechanical energy varies for a subject from bodily location to bodily location and from time to time because we adapt to mechanical energy. This may seem a straightforward matter, when we adapt positively the damage threshold goes up and when we adapt negatively it goes down. But there is more to adaptation than this. The first issue to consider is that there is a sense in which tissues are constantly being damaged as part of the process of breakdown and repair. The second is to question whether adaptive changes to a tissue affect the damage threshold of the individual (micro-) constituents of that tissue – the thicker a rope the stronger it is because a given mechanical force is distributed between more fibres, there is no implication that the damage threshold of individual strands of the rope changes. Finally, there are reasons to believe that changes in the relationship between intensities of mechanical energy and the damage threshold in undamaged tissue make little difference to the functioning of *FS*, because

[35] This is an oversimplification. A thermode (a scientific instrument used to deliver precisely controlled intensities of thermal energy) might be calibrated to deliver thermal energy at 40°C, but the temperature at the sensory receptor is a function of the skin-depth of the receptor, the length of time that the thermode is applied and the dissipation of heat by the tissues. The latter is a variable involving local circulation. The anatomy and physiology of capillaries, arterioles, venules etc. is adaptable. Nevertheless, it remains the case that skin and other tissues become damaged when the temperature *in the tissues* reaches approximately 47°C.

we do not normally respond to at least some of the intensities of mechanical energy that exceed the noxious threshold.

In the interest of clarity, in this section I will take it that the noxious threshold is an objective threshold marking the lower limit of threat. Additionally, in contradiction of the arguments in the preceding sections, I take it that all the nociceptors expressed by nociceptive neurons have thresholds at or above the noxious threshold. So nociceptors cannot transduce intensities of energy that are below the noxious threshold.

Almost all types of tissue are being constantly *regenerated* through processes of breakdown and rebuilding. The rate and balance between these processes is affected by the intensity and repetition of mechanical forces, combined with rest and the capacities of any given subject's tissues at a given time. So in this regenerative sense most tissues are constantly damaged and mechanical energy at many bodily locations is having a damaging effect.[36] Regenerative processes are essential factors in a tissue's ability to *adapt* to mechanical energy. The most obvious example of the positive effect of these processes is an increase in the ability to cope with and generate higher intensities of mechanical energy following a weight-training programme. Increased activity leads to an increase in the rate and extent of tissue breakdown and rebuilding. If rest periods are adequate but not excessive in length, then tissues like bone, muscle, tendon, ligament and skin increase their ability to cope with mechanical energy.

[36] It is worth adding that this is one of the main reasons why the concept of undamaged or normal tissue is difficult to apply in real life.

Although the term 'noxious' is not conventionally used to refer to the enhanced breakdown of tissue in response to the mechanical energy involved in the normal regenerative process, this is a vague conceptual distinction. For a given subject, identical intensities and repetitions of mechanical energy at a bodily location can lead to adaptation over time, but if rest periods are too short (i.e. the same amount of work being done over less time) breakdown can exceed rebuilding resulting in frank damage to the tissues. In this way relatively low intensities of mechanical energy can be constituents in noxious events (e.g., typing five days a week for many years can cause tendon damage).[37] At the other extreme a high intensity of mechanical energy can directly damage tissue without affecting the balance between breakdown and rebuilding (e.g. a runner who sprains an ankle ligament when she puts her foot in a pothole). As almost any intensity of mechanical energy might be a constituent of a noxious event, it follows that almost any intensity of mechanical energy is potentially relevant to the nocifensive function of *FS*.

For the purpose of my analysis, the variability of the noxious threshold for mechanical energy in undamaged tissue can be summarised in this way:

a) For a given subject a particular intensity of mechanical energy might well be a constituent of a noxious event at one bodily location but not another at a given time.

[37] For more on the damaging of bodily tissues by low intensity mechanical energy, see Lynn 2006.

b) For a given subject a particular intensity of mechanical energy might be a constituent of a noxious event at a given bodily location at one time but not another. [38]

The summaries in (*a*) and (*b*) express the fact that the strength of muscle, tendon, ligament, bone etc. increases (or decreases) with the right (wrong) sort of activity as a consequence of the ability to adapt. These increases (or decreases) in strength just are increases (and decreases) in the capacity to cope with mechanical energy.[39] Hence, they correspond to increases and decreases in the noxious threshold of these tissues. But as the rope illustration in the opening paragraph of this sub-section shows, an increase in the damage threshold of a tissue as a whole does not imply an increase in the constituents of that tissue.

Muscles are constituted by muscle fibres arranged into bundles called 'motor units'. Motor units are innervated by a single motor (efferent) neuron, and each muscle is a functional unit with the capacity to contract and relax a proportion of its motor units. As the motor system is only able to recruit a proportion of the motor units in a given muscle at any one time, the ability to generate higher intensities of mechanical energy is restricted by the ability to recruit motor units. This proportion increases in response to training (Proske and Morgan 2001). So, for all that has been said so far, it might be that the damage threshold of the muscle as a whole increases when a greater

[38] In addition, (*c*) at a given time a particular intensity of mechanical energy might be a constituent of a noxious event involving one subject's but not another's right anterior cruciate ligament (say). By (*a*)-(*c*) whether or not and to what degree an intensity of mechanical energy is noxious is relative to bodily location for a subject, to time for a subject, and from subject to subject. These are very good reasons to reject the dispositional interpretation of a 'noxious stimulus' (*E2*) I discussed in section *4.2.2*.

[39] Muscle tissue is a little different because 'strength' usually refers to the ability to generate rather than absorb mechanical energy, but it is reasonable to assume an increase or decrease in the ability to generate mechanical energy goes hand in hand with an increase or decrease in the noxious threshold.

proportion of motor units share a given amount of work (i.e. each motor unit is doing less work),[40] and the damage threshold decreases when a smaller proportion of motor units share a given amount of work (i.e. each motor unit is doing more work because the subject has become less able to recruit motor units). In this way, there is no change to the damage threshold of each motor unit despite changes in the damage threshold (and the ability to do work) of the muscle as a whole. As sensory receptors are located in motor units, there is no change in the relationship between the nociceptive threshold and the damage threshold of the motor unit.

Although this is persuasive as it stands, the reasoning is based on a partial account of muscle biology. Muscle fibres do not have the ability to contract more or less strongly, they can only contract or relax. A motor unit has the same contractile characteristics as the sum of its constituent fibres; all its constituent muscle fibres are either contracting maximally or relaxing. But the muscle fibres within a motor unit do become stronger or weaker through adaptive change.[41] In this way, when the fibres in the motor units of a muscle become stronger the lifting of a 25kg weight at a given speed requires the recruitment of 40% (say) of the motor units within a muscle, whereas the same task used to require the recruitment of 65% (say) of the motor units within that muscle. As the fibres that constitute motor units and muscles increase and decrease in strength the noxious thresholds of a muscle fibre, a motor unit and a muscle also increase and decrease. If *FS* is to discriminate noxious intensities of mechanical energy in muscle tissue then either nociceptive thresholds vary with the

[40] 'Work' is intended to convey the generation of mechanical energy.
[41] Muscle fibres hypertrophy (they increase their cross-sectional area) in response to training. This enables a fibre to generate greater force. Conversely, a muscle fibre that is not recruited sufficiently often, decreases in cross-sectional area and loses power.

adaptive changes in muscle or CNS structures of *FS* are involved in a complex evaluative process that weighs supply against demand. I will question whether *FS* does discriminate noxious intensities of mechanical energy after briefly considering variation in nociceptive thresholds and the claim that an evaluative process would need to be complex.

Although there is overwhelming evidence that nociceptive thresholds fall in response to the release of algogenic substances like prostaglandins and nerve growth factor from *damaged* tissue (Coutaux et al, 2005),[42] no equivalent mechanism seems to change these thresholds in undamaged tissue. Of course, given the remarks I made above the concept of undamaged tissue is an oversimplification. But this matter is not relevant here. The process of regeneration is ongoing so if algogenic substances are released into the tissues the levels are unlikely to fluctuate significantly. If the fluctuations are significant, then algogenic substances would be high post-exercise so the nociceptive thresholds would be low post-exercise and return to their previous level thereafter. If receptor plasticity is to more or less fix the relationship between nociceptive and noxious thresholds, longer term changes are needed; short term changes like this are not relevant.

Although I have written of fixing the relationship between nociceptive and noxious thresholds, it can be argued that a close relationship between these thresholds is not required for *FS* to fulfil its nocifensive function. Injury-prevention is all that is

[42] This includes a proportion of the total population of C-fibres which express 'silent nociceptors'. Silent nociceptors have stimulus thresholds well above the damage threshold of tissues. It is thought that the action of 'algogenic' substances reduces these stimulus thresholds at times of injury so that there is a massive increase in the barrage of action potentials to the nocifensive system in response to thermal, mechanical and chemical stimuli. See Schmidt et al, (1995) for more on silent nociceptors.

required and the function of preventing injury is served by over-protection; if the nociceptive threshold is at or below the noxious threshold of the tissues, *FS* can function advantageously.

Once again this *may* be true in the presence of tissue damage. When there is frank damage, particularly in the early (acute) stages, algogenic substances are released and the stimulus thresholds of neurons fall. This fairly crude adaptive mechanism is evident in *Sprain* (see chapter 1, section *1.2.5*). When the subject rises from a chair she feels quite intense *TYP*. This is an example of over-protection because *TYP* eases considerably as she moves around. However, the fact that her *TYP* eases is suggestive that her *TYP* is not the product of a crude mechanism. If it were a simple mechanism *TYP* would not moderate to such an extent.[43] Furthermore, an explanation like this would not account for the changes in the response profiles of nociceptors in response to the adaptation of undamaged tissues that seems to be required to sustain the position that noxious intensities of energy are discriminated peripherally.

I have argued here that the damage thresholds of tissues change as a consequence of adaptive changes. I have also argued that no peripheral mechanism can effect changes in nociceptors so that their stimulus thresholds reflect these changes in the damage threshold. The issue I have not addressed is whether changes to the damage thresholds of undamaged tissue are relevant to *FS*.

[43] I discuss *Sprain* in chapter 6, section *6.3.3*.

FS receives input from mechanically sensitive neurons. The evidence for this is provided by *Sprain* and other examples in which mechanical forces are imposed on damaged tissue. So *FS* responds to intensities of mechanical energy in the presence of tissue damage. It also responds to the compression of tissues because pinching skin or other tissues usually results in an experience of *TYP*. But the tissues do not really adapt to compression forces so these responses are uninformative. The discrimination of noxious intensities of energy fulfils a much less clear-cut role in the prevention of injury to undamaged tissue by other types of mechanical force.

The evidence that *FS* does not make these discriminations is that the *occurrence* of injury as a consequence of both sudden high intensity mechanical energy and repetitive low intensity energy is often not accompanied by *TYP*. There are at least four issues here. The first concerns the difficulty of determining whether a particular intensity of energy is noxious. When we run for a bus, we generate and absorb high intensities of energy. Are these intensities close to or above the noxious threshold? If not, do they exceed the noxious threshold when we run for a mile or more? It is difficult to answer these questions with any kind of certainty. One of the variables concerns what we normally do. If you normally do little activity, the answer *may* be yes to the first question. If you normally run much longer distances the answer *may* be no to both questions. Perhaps *FS* does not have the resources to make discriminations in such circumstances so it does not discriminate noxious intensities of mechanical energy except when it involves compression (crushing, pinching, etc.) because we cannot adapt to compression forces.

The second is that the events being discussed involve energy that is being generated by muscles. *FS* may not respond because nocifensive responses to high intensities of energy that might be noxious (but not damaging) might seriously inhibit our ability to move balance and so on. Furthermore, sub-personal systems control this energy so these systems may well inhibit the relevant neurophysiological activity as the intensities of energy approximate the noxious threshold. In other words, *FS* may be responding in subtle ways by altering muscle tone rather than by evoking *TYP*.

Third, there is a sense in which a subject who is generating mechanical energy at a certain intensity must be adapted to that energy (if she were not she would not be able to generate that intensity), therefore she is unlikely to be damaged by this intensity.[44]

Fourth, sudden high intensities of mechanical energy may be irresistible; i.e. the reason subjects often do not experience *TYP* in response to energy that ruptures a tendon or produces a gunshot wound is that *TYP* cannot alter the outcomes.[45]

These considerations make it difficult to draw firm conclusions about the effects of adaptation on discrimination. Viewed mechanistically it is most likely that when the subject in *Sprain* experiences *TYP, FS* is responding to a significant increase in the frequency of action potentials (an increase in the afferent barrage). So it is reasonable

[44] I say "there is a sense in which…" because this is a complex issue. The generation of energy involves muscles and tendons because muscles generate the energy and tendons attach muscle to bone. Bone transmits the energy. So each of these tissues adapts to increased demand. As far as adaptation is concerned, the effect of increased demand on ligaments is less clear. Ligaments give non-contractile support to joints. I assume that increased demand on muscle etc. also increases the demand on ligaments. Additionally, there is the problem posed by the effects of repetition and by the absorption of external forces (like gravity). These details are not really of concern. My point is that *FS* may not evoke *TYP* in response to some relevant episodes involving mechanical energy, because there is no advantage to such a response.

[45] See the Reagan case in chapter 1, section *1.2.5*. For further discussion see chapter 6, section *6.3.2*.

to assume that *FS* would also respond to a significant increase in afferent barrage from neurons that innervate undamaged tissue. The question is whether the afferent barrage increases significantly when the mechanical noxious threshold in undamaged tissue is reached. There are empirical reasons (the ruptured tendon and gunshot examples) to think it might not. But on balance my view is that if *FS* has the resources to discriminate noxious mechanical energy in damaged tissue then it has the resources to do so in undamaged tissue. If it does not have such resources, my claim that CNS structures of *FS* fulfil a significant role in the discrimination of noxious intensities of energy still stands because I have argued that sensory receptors cannot discriminate that intensities of energy are noxious. At the very least, these CNS structures are involved in a simple process of discriminating which patterns of impulses represent intensities of energy that may be threatening to the tissues. Consideration of the changes in damage threshold in the presence of tissue damage and the variability of *TYP* in cases like *Sprain* strongly suggest that the discriminatory process conducted by *FS* is complex rather than a straightforward gating mechanism.

4.5 NOCICEPTIVE CONCEPTS – USEFUL TOOLS OR IMPEDIMENTS

Where do these arguments leave nociceptive concepts? In section *4.3*, I provided revisionary definitions of a nociceptor and a nociceptive neuron (*NR′* and *NN*). These definitions are consistent with the arguments I have conducted above. For all that I have discussed it may be that some Aβ-neurons are constituents of *FS* because the impulse patterns generated by these fibres are relevant to *FS*, at least at times. These

'first order' neurons also synapse with wide dynamic range (WDR) neurons[46] of the dorsal horn of the spinal cord. These WDR neurons also receive input from the sub-population of primary afferent Aδ- and C-fibres that are classified as 'nociceptive' so there is anatomical evidence that input from these low threshold Aβ-neurons is utilised by *FS*. So the receptors expressed by these Aβ-neurons are nociceptors by *NR'* and they are nociceptive neurons by *NN*. The trouble with this categorisation is that it suggests a specificity that may not be justified by the biological facts.[47] The conception of *FS* I will develop in chapter 5 section *5.6* leaves open the possibility that peripheral neurons are not specific to a particular functional system and the account I give of the perceptual features of *Q* (in chapter 5, section *5.1.1*) requires that some neurons that are constituents of different perceptual systems are also constituents of *FS*. Hence, it may well be that these Aβ-neurons fulfil functional roles in other systems. The labelling of them and the sensory receptors they express as nociceptive neurons and nociceptors respectively is somewhat misleading in this light.

In support of this lack of functional specificity, consider that a broad range of mechanical energy events that in total involve all the body tissues are constantly occurring. Many of the intensities of energy involved in these events are being transduced by sensory receptors. Given the potentially damaging effects of repetitive intensities of mechanical energy it is possible that almost any of these events involves energy that could be having a noxious effect. However, it is unlikely that any one of these events is noxious at a given time. Therefore, sensory receptors throughout the body are transducing intensities of energy that *could be* but are probably not

[46] I introduced WDR neurons at the end of section *4.4.1*, this chapter.
[47] This mirrors the concerns that Ianetti and Mouraux have about the pain matrix (see chapter 3, section *3.2*)

constituents of noxious events. These sensory receptors are expressed by neurons that are constituents of systems that have functions including motor control, proprioception and the production of conscious sensations (like touch, pressure and stretch). If neurons are specific to *FS*, then these constituents of systems other than *FS* are not constituents of *FS*.

It might be claimed that this is not as biologically profligate as it sounds because the specificity of the peripheral system provides an advantage. The question is what advantage. While it is true that sensory neurons are anatomically and physiologically complex, functionally they can be conceived simply in terms of information about energy. As I have been at pains to argue there is no fundamental difference between the information provided by the putative class 'nociceptive neuron' and any other class of sensory neuron. The difference is in the functional systems that utilise the information. This determines the way the information is interpreted. Why would organisms evolve separate peripheral systems that reproduce information about mechanical energy rather than divergent systems that disseminate relevant peripheral discriminations across functional systems? It seems implausible to me that they would.

In the absence of an argument to the contrary, the position that the mechanically sensitive neurons which provide input to *FS* also provide input to other systems is consistent with the evidence and arguments I have presented in this and preceding chapters. The implication is that a significant proportion (perhaps the vast majority) of putative nociceptive neurons are also neurons with functions other than nocifense (e.g.

perceptual, motor and proprioceptive functions). In which case there is no difference between many (most?) nociceptive neurons and other categories of neuron. Consequently, the concepts of a nociceptor and a nociceptive neuron are misleading

Although Craig has written that the category 'nociceptors' is heuristically of enormous value (2002, p.657), I disagree. The focus of scientists on intensities of thermal energy that are close to the damage threshold for undamaged tissue has lead to the belief that a class of sensory neuron has the capacity to discriminate noxious intensities of energy (if not noxious stimuli). It may well be that some neurons are specific to *FS* but it is a mistake to assume the existence of a complete sensory system on this basis. This assumption has closed not only conceptual space but also minds to the possibility that CNS structures of *FS* are significantly involved in the discrimination of noxious intensities of energy.

5 MOTIVATIONALISM ABOUT PAIN

In previous chapters, I have identified the typical experiences we identify as pain (*TYP*) as an output of a system that has a nocifensive function (*FS*). This system functions to prevent harm and promote recovery from harm. As such, *FS* must have a capacity for discriminating intensities of energy that could damage tissue. One of the insights provided by the arguments of chapter 4 is that most intensities of mechanical energy could damage either undamaged or damaged tissue, and some intensities of thermal energy that could not damage undamaged tissue have the potential to further damage damaged tissue. Hence, many (perhaps most) intensities of energy are relevant for nocifense, and so thermally and mechanically sensitive sensory neurons with a wide range of stimulus thresholds provide input to *FS*. It is extremely doubtful that these inputs are specific to *FS* as information provided by thermally and mechanically sensitive sensory neurons with a wide range of stimulus thresholds are also relevant to functional systems other than *FS*. So it is reasonable to assume on the grounds of economy that information generated by peripheral neurons is available to *FS* and other functional systems.[1] Discrimination between inputs that do or do not require a nocifensive response is a function of central nervous system structures of *FS*. This chapter is concerned with the impact of these matters and the development of the motivational thesis I call 'near-motivationalism'.

[1] I discuss this in chapter 4 section *4.5*, and in this chapter section *5.6*.

In section *5.1*, I recap and clarify the commitments of motivationalism. In doing so I present the view that mental states can have more than one function. This provides an explanation for the problem posed to motivationalism by the apparently perceptual features of pain (P1). In sub-section *5.1.1*, I discuss the subjective character of *Q* concluding that tokens of *Q* are not specific to pain. In section *5.2*, I take this conclusion in contribution to the resolution of the question of whether pain has proprietary phenomenology and in contradiction of the claim that unpleasantness is not necessary for pain (section *5.3*). These arguments solve two of the problems associated with P2. In section *5.4*, I analyse the unpleasantness of pain, taking the position *U* adds negative affective tone to tokens of *Q*. It is this combination that we usually recognise as pain. In section *5.5*, I develop my claim that pain functions to motivate injury-preventing behaviour. Finally in section *5.6*, I provide an account of *FS* and explain how *Q* and *U* both function as motivational mental states as constituents of pain to motivate nocifensive behaviours (this is a solution to P3) and resist the challenge that near-motivationalism is just a version of the mixed position.

5.1 THE PERCEPTUAL FEATURES OF Q

The claim that *TYP* or any other experience has a particular function needs clarification. Human cognitive abilities are such that pain and other experiences can be used for all manner of functional purposes – for example, I use *TYP* as a means of introspecting conscious experience – but this does not mean that the function of *TYP* is introspection. In my terms, this sort of learned use is an example of *ontogenetic utility*. Functions like this have had no bearing on the nature of the experience or its

evolved utility. By contrast, experiences have *primary functions*. This is reflected in the observation that the ability to experience in a particular way came to exist for this function. An experience *may* also have a *secondary function*, which is reflected in the evolved ability to utilise experiences that have evolved for a primary purpose in another functional role. The claim that *TYP is* motivational is a claim about the primary function of *TYP*. In more detail, it is the claim that the ability to have a composite experience constituted by *Q* and *U* has evolved for the purpose of motivating injury-preventing behaviour. But this need not be the case for the individual components of *TYP* (*Q* and *U*). The affectively neutral phenomenal qualities that constitute *Q* (say) may fulfil a secondary function in *TYP*. To be clear on this, what matters is the primary function of *TYP* and the functions that *Q* and *U* fulfil as constituents of *TYP*. The sum total of the relevant functions of *Q* and *U* (i.e. their individual functional contributions to the overall function of *TYP*) must add up to the primary function of *TYP*. Motivationalism then, is the thesis that both *Q* and *U* fulfil motivational functions *as constituents of the composite experience TYP*.

Although I have rejected Colin Klein's version of motivationalism[2] both of us face the obstacle of explaining the *prima facie* evidence that tokens of *Q* are perceptual; i.e. the varied qualities, intensities and locations of *Q*. I begin my alternative motivational account 'near-motivationalism' by considering the primary and secondary functions of *Q*.

[2] I reject his position in chapter 2, section *2.4*.

5.1.1 The nature of Q

The proposition that the ability to experience *Q* has evolved to fulfil a *primary* motivational role in *FS* may be true but it is difficult to make a persuasive case for its truth. *Q* has the hallmarks of an experience that is primarily perceptual. It varies phenomenally, in intensity and in felt location just like the mechanical sensations we describe in terms of touch and pressure. Furthermore, science associates *Q* with the somatosensory cortex, an area of the brain that is linked with sensory discrimination. The question is whether *Q* fulfils this primary function as a constituent of pain.

One reason for doubt is that the experiences of *TYP* we get in response to thermal and mechanical stimuli are easily distinguished by their subjects. This strongly suggests that the respective phenomenal qualities are drawn from different quality spaces.[3] Given that the primary function of *TYP* is injury-preventing behaviour, if the primary perceptual function of tokens of *Q* is a functional component of *TYP*, it is reasonable to assume that the ability to distinguish thermal and mechanical *TYP*s would be advantageous. What might that advantage be? The only thing that comes to mind is that noxious thermal and mechanical energy require different injury-preventing behaviours. But it does not seem to me that there is any significant difference in the behaviours we exhibit in response to these different types of energy; we avoid, withdraw and so on.[4] If there is no advantage, the best (perhaps the only) explanation for the subjective difference between thermal and mechanical *TYP*s is that tokens of *Q*

[3] My use of 'quality space' commits me to nothing more than the position that visual experiences (say) are subserved by a repository of phenomenal qualities that are united by their similarity. In particular, I am not committed to the view that quality spaces correspond with sensory modalities. I briefly discuss these issues in this section.

[4] I discuss this in more detail in the next section.

fulfil a primary perceptual role in some functional system other than *FS*. If this is right, *Q* fulfils a secondary function in *TYP*. For all that has been said so far there is no obstacle to the claim that this secondary function is motivational

There is a more complex possibility. A progenitor of the ability to generate *Q* evolved in response to nocifensive demands. This species then evolved the ability to utilise tokens of *Q* for another (secondary) purpose. The phylogenetic pressures associated with this secondary function then refined the subjective features of *Q*. These features were not necessary for its primary (original) nocifensive function. In this way, *Q* could have features that play no functional role in *TYP* even though this is its primary role. Fortunately, I have no need to settle this complex matter, either way I get a plausible explanation for the perceptual features of *Q* that is consistent with motivationalism. In what follows I will simply assume the former; the subjective nature of *Q* is derived from a primary perceptual function so there is no obvious obstacle to the claim that tokens of *Q* fulfil a (secondary) motivational function as constituents of *TYP*.

The purpose of this section has been to present an account that rationalises the (subjective) perceptual features of *Q* with a motivational function as a constituent of *TYP*. The purpose has not been to argue that *Q* has a perceptual function as a constituent of a functional system other than *FS* and a motivational function in *TYP*. This argument will drop out of subsequent sections. It remains to point out that if *Q* has a function other than as a constituent of *TYP*, then the phenomenology of *Q* is not unique to *TYP*. This conditional statement would be of less consequence for those

who assume that *TYP* is characterised by proprietary phenomenology if there were overwhelming evidence that *FS* receives inputs from a specific (nociceptive) sensory system, but as I argued in chapter 4, *TYP* is not subserved by such a system. As the claim that *Q* does not contribute unique phenomenology to *TYP* is of considerable significance for my thesis,[5] I will provide an independent argument that *TYP* lacks proprietary phenomenology.

5.2 DOES PAIN HAVE PROPRIETARY PHENOMENOLOGY?

In chapter 1 section *1.2.2*, I introduced a term 'proprietary phenomenology' that is not commonly used in the literature. In this section, I am not taking 'proprietary phenomenology' to refer to a unique combination of subjective experiences. If I were then *TYP* might well have proprietary phenomenology because it is uniquely constituted by *Q* and *U*. Here 'proprietary phenomenology' is intended to reflect ownership of a quality space; e.g., visual experience is subserved by its own quality space. If *TYP* has its own quality space(s), then either or both *Q* and *U* are constituted by qualities drawn from that space (those spaces). This section is focused on *Q* so I am taking it that if *TYP* has propoprietary phenomenology that phenomenology is derived from tokens of *Q* (the affectively neutral component of *TYP*). I will consider the nature of *U* in section *5.4*.

[5] If *Q* contributes unique phenomenal character to *TYP*, then it is most likely that *TYP* is subserved by a specific sensory system. In which case I have no more resources available to explain the problem cases expressed in P4-P6 than other theorists.

The arguments I have presented in chapter 4, that sensory input to *FS* is not specific to nocifense and doubts surrounding the functional difference between thermal and mechanical *TYP*s provide good reasons to be suspicious of the claim that *TYP* has proprietary phenomenology, but they are not conclusive. For all I have said *Q* may not have a secondary function. But on a standard characterisation of quality space these arguments are enough to reject the claim that *TYP* has proprietary phenomenology. Here is Austen Clark:

> A *quality space* is an ordering of the qualities presented by a sensory modality in which relative similarities among those qualities are represented by their relative distances. Qualities that are relatively similar to one another are closer to one another than are qualities that are relatively less similar (2000, p.4; author's italics).[6]

The position that has emerged as a consequence of the arguments I presented in chapter 4 is that no sensory system is specific to *TYP*. This amounts to the position that *TYP* is not a sensory modality. So by Clark's conception of quality space there is no quality space for *TYP*.

There are three good reasons to reject this argument. First, it requires a robust theory of sensory modality. Although sensory modalities are conventionally associated with types of stimulus (hence, my claim that *TYP* is not a sensory modality). I am not aware of any *robust* theory. Part of the problem is that the concept of 'stimulus type'

[6] See also Rosenthal (2005 pp196-198).

is of itself problematic. My analysis of a noxious stimulus exemplifies this problem. Another is that sensory input is integrated in the CNS. This complicates the concept of a sensory modality and this in turn weakens the concept of a quality space.

Second, the standard conception of quality space is in my view seriously flawed in other ways. For example, the relative distance within a quality space is claimed to be a function of the organisation of mind-independent qualities. In other words, the structure of a quality space is claimed to be determined by the structural organisation of mind-independent properties (Clark, 2000; Rosenthal, 2005). Certainly, consideration of the visible spectrum pumps the intuition that the mind-independent world is spatially organised in the way required. But sodium chloride (salt), ascorbic acid (vinegar), and compounds of carbon, hydrogen and oxygen (sugars) have mind-independent properties that are detectable by a sensory modality. It seems implausible that these substances are spatially organised independently of perceivers in some way that justifies the claim that the distance between salty, sour and sugary tastes represents an equivalent mind-independent structure. Additionally, the relationship between wavelengths of light and colour experience predicts that there is a greater contrast *for the subject* between phenomenal red and phenomenal violet than between phenomenal red and phenomenal yellow. But my experience at least, is that red does not seem more like yellow than violet.

Third, even if there were no such difficulties, sensory neurology is being used as a means of individuating types of phenomenal experience. A *bottom-up* (sensory nerve-ending to brain) approach to the classification of *subjective* experience is

inappropriate. By contrast, a top-down approach to phenomenal experience is apposite because the *explanandum* is subjective experience. A green experience is distinct from a blue experience, but there is also a similarity between these experiences that is not shared by a green experience and a buzzing sound. The question of whether *TYP*, vision, audition or any other putative types of qualitative experience have proprietary phenomenology can be answered by consideration of the differences between token phenomenal experiences.

It is possible (for a subject) to make a *gradual transition* between *any* two token colour experiences (say). There is a gradual subjective transition from red through orange to yellow. This is to be contrasted with the impossibility of a gradual transition between the phenomenal qualities that constitute a buzzing sound, a green experience, a touch sensation and the smell of coffee. There are phenomenal '*hard edges*' between these experiences that enable us to distinguish auditory, visual, mechanical and olfactory experiences. But this does not mean that, in the absence of hard edges, we would not be able distinguish these experiences; paradigm examples of each would be distinguishable just as we can distinguish between loud, moderate and soft sounds or red and yellow colours. Rather the implication of hard edges is that there are no marginal cases. There is no mistaking a visual experience for an auditory experience.

Hard edges delimit distinct types of phenomenology. If this is not taken to be the case then either distinctions between types of phenomenology are arbitrary or we are basing distinctions on something other than the experiences themselves; sensory

neurology perhaps. Neither would be an appropriate way of individuating types of phenomenology. So the question of whether *TYP* has proprietary phenomenology can be answered by consideration of whether or not there is a hard edge between the phenomenology of *TYP* and the phenomenology of other experiences. Given the remarks I made about the nature of *U* at the beginning of this section, the question of whether *TYP* has hard edges is the same as the question of whether tokens of *Q* are constituents of any other type of experience. If *TYP* has hard edges then *Q* is not a constituent of any other type of experience and *vice versa*. It is *not* equivalent to the question of whether *Q* has hard edges. *Q must* have hard edges because it is a type (or types) of phenomenology. This does not imply that *TYP* has proprietary phenomenology because *Q* may constitute or be a constituent of experiences other than *TYP*.

The trouble is that the composite structure of *TYP* raises a practical difficulty. The difficulty is that an apparent hard edge between the phenomenal component of *TYP* and another phenomenal experience may be nothing more than an appearance caused by the attaching of *U* to a token of *Q*. Despite this worry, the following is telling:

Stretch When you begin to gently stretch your finger back into extension you feel a slight stretching (mechanical) sensation. An increase in the intensity of the stretch correlates with an increase in the intensity of the stretching sensation. At some point (1), which is difficult to precisely determine, the sensation

> begins to become unpleasant. As you continue to increase the stretch the motivation to stop will also increase.[7]

By definition, the onset of *TYP* coincides with 1 (because this is the point at which *U* is attached to a token of *Q*). On the position that *TYP* has proprietary phenomenology, prior to 1 you are experiencing a phenomenal quality that is not a token of *Q* (call this a token of '*R*'); at 1 *R* is replaced by both *Q* and *U*. Consequently, your inability to precisely determine the point at which *R* is replaced by *TYP* is remarkable. Contrast this with the position that *TYP* lacks proprietary phenomenology. Prior to 1 you are experiencing a token of *Q*. As the intensity of the stretch increases the intensity of *Q* increases so that immediately pre- and at 1 the difference between the intensities of *Q* is very slight. At 1, the attachment of a *very* low intensity of *U* makes little difference to *Q*. This explains your inability to precisely determine the transition from *Q* to *TYP*.

In addition to this argument, I can appeal to biological economy – the ability to switch from phenomenal qualities of one type to another at 1 seems unnecessarily profligate as it requires two types of phenomenal quality and a switching mechanism that turns one phenomenal quality off and another on – but in my view the preceding arguments are persuasive enough. *FS* is not subserved by a specific sensory system, there seems no good functional reason for the ability to experience thermal and mechanical *TYP*s, and there is no hard edge between *TYP* and the phenomenology associated with thermal and mechanical stimuli.

[7] Although *Stretch* concerns mechanical stimuli, a similar example can be constructed for thermal stimuli.

These arguments count strongly against the position that the characteristic feel of *TYP* is given by *Q*, but might *U* contribute the unique phenomenology to *TYP*; i.e. *U* is constituted by phenomenal qualities that are specific to pain. My arguments that no sensory system is specific to *TYP* counts against this possibility, but the problem posed by thermal and mechanical *TYP*s would not be a problem; if *U* provides unique phenomenology, the need for an explanation of the functional role of thermal and mechanical *TYP*s disappears. The difference between thermal and mechanical qualities of *Q* fulfils a function in a system(s) other than *FS*. It fulfils no function in *FS*. The inability to precisely determine when *U* is evoked can be explained by its extremely low intensity at 1 (see *Stretch*, above); *U* is barely discernible at these intensities hence our inability. The possibility that *U* is a phenomenal experience unique to *TYP*,[8] is discussed in section *5.4*.

5.3 IS UNPLEASANTNESS NECESSARY FOR PAIN?

Several philosophers claim that an experience of *Q* is sufficient for pain.[9] Although few provide much argument all these philosophers base their positions on the reports of subjects, like those with asymbolia, who say they are experiencing pain that is not unpleasant.[10] These reports, they urge, should be taken at face value – that is reports

[8] A caveat is needed to account for putative cases where *U* is experienced in the absence of *Q*. Nikolas Grahek cites a single case study that may be evidence of just such an experience (Grahek, 2007, pp.95-140). See below.
[9] I have in mind philosophers who otherwise have quite different stances on pain. For example, Bain (2014), Grahek (2007), Hall (1989), and Klein (forthcoming).
[10] Grahek (2007) and Hall (1989) are notable exceptions; both provide detailed arguments.

of pain are a powerful reason to accept that their subjects are in pain. A typical argument for this position goes like this:[11]

1) Premise: The recognitional concept of pain: Pains are the experiences that could in principle be identified as pain under *REC*.[12]
2) Conditional: Given 'premise' (1), if a subject were to identify an experience as pain under *REC*, and report that the experience is not unpleasant then that experience would be a pain.
3) Evidence: There are actual cases like those expressed in 'Conditional' (2).
4) Conclusion: Pains are not necessarily unpleasant.

As a general rule, the argumentative emphasis has been on 'evidence'; there are certainly reasons to be suspicious of the claims that the reports of those being treated with morphine, lobotomised patients and those with asymbolia are consistent with *REC*. But on balance the evidence weighs in favour of the claim that these subjects are identifying affectively neutral experiences as pain under *REC*.[13] Much less attention has been devoted to 'premise'.

'Premise' is underpinned by the assumption that subjects are best placed to judge whether they are in pain because they are able to recognise something, a common subjective core, that unites the experiences we could identify as pain under *REC*. There are only three possible candidates for this common core: affectively neutral

[11] This argument is derived from Hall's 1989 paper, 'Pains are not necessarily unpleasant'.
[12] I introduced *REC* in chapter 1, section *1.1*: A subject holds *REC* if and only if she is able to categorise an occurrent experience as pain by reference to her memories of experiences she has learned to categorise together as pain.
[13] I discuss these issues in chapter 1, section *1.2.2*.

phenomenal qualities (*Q*), the affective mental state that makes it feel as if *Q* is intrinsically unpleasant (*U*) or affectively neutral phenomenal qualities that feel as if they are intrinsically unpleasant (*TYP*).[14] The latter two are ruled out by asymbolia so affectively neutral phenomenology has the common core that unites experiences under the label pain. This is not a strong argument as it stands. Nothing rules out the possibility that the phenomenal core is a motley collection of qualities we have learned to recognise by association with tissue damaging events. In which case pain would be defined as something of this sort "A sensory experience associated with actual or potential tissue damage or described in terms of such damage."[15]

In my view, a definition like this would trivialise the concept of pain. The original title of Paul Brand and Philip Yancey's 1993 book, Pain: The Gift Nobody Wants,[16] reflects the dichotomy that lies at the heart of both scientific and commonsense conceptions of pain. Pain fulfils an essential functional role and yet pain also has the power to cause suffering. If we accept that affectively neutral phenomenal qualities are sufficient for pain we are stripping pain of its functional and cultural significance. In the absence of unpleasantness pains are relatively trivial experiences. This is a good reason to reject the claim that pains are not necessarily unpleasant.

The obvious response to this objection is to gesture towards the concept of a nociceptive system as evidence that the phenomenal qualities of pain have a *unique*

[14] These are the only candidates because the subjective character of a token of *TYP* is exhausted by an affectively neutral phenomenal quality that feels as if it is intrinsically unpleasant.
[15] This suggestion is derived from the IASP's definition of pain: "An unpleasant sensory and emotional experience associated with actual or potential tissue damage or defined in terms of such damage" (2014). Note that by the IASP's definition *all* pains are unpleasant.
[16] Its new title "The Gift of Pain" is much less compelling.

character that subserves the detection of noxious intensities of energy. The issue then is one of whether or not we *should* take 'pain' to be the label for tokens of this unique phenomenology. Bain, Grahek, Hall and Klein all believe we should but I do not think this response is strong enough to resist the triviality objection I raised above. 'Pain' refers to an experience that fulfils an important function. Unpleasantness is a vital component of this function. Affectively neutral 'pains' lack this importance. In order to avoid trivialising the concept of pain it would be better to deny that asymbolia pain is pain.

This is a good reason to reject 'premise', but the argument of the preceding section is much stronger. *TYP* is not subserved by a specific sensory system and tokens of *Q* are not uniquely for *TYP*. The phenomenal qualities which constitute the affectively neutral component of *TYP* are tokens of the thermal or mechanical phenomenal qualities that also subserve innocuous sensations. So the affectively neutral phenomenal qualities of *TYP* have a common subjective core – they are tokens of either thermal or mechanical phenomenology (they are tokens of *Q*) – but they share this core with *all* tokens of *Q*. 'Pain' is not an appropriate term for all tokens of *Q*, because some tokens do not fulfil an injury-preventing function in *FS*. And as there is no subjective difference between the tokens of *Q* that fulfil different functions 'pain' is not an appropriate term for any tokens of *Q*. A token of *Q* must be experienced as if it were intrinsically unpleasant in order to fulfil its injury-preventing function, and a token of *Q* that is being utilised by *FS* for injury-preventing purposes can only be identified as such when it is experienced as if it were intrinsically unpleasant.[17]

[17] The "pain" of asymbolia is not a counterexample to this pair of claims even though these "pains" could be involved in cognitive processes that have the potential to yield injury-preventing behaviour.

Hence, pains are necessarily unpleasant ('pain' is synonymous with '*TYP*'). As this is a strong argument, I will drop the neutral term '*TYP*'. From this point in my thesis 'pain' refers to composite experiences constituted by tokens of thermal and mechanical phenomenal qualities and a further mental state (*U*) that makes it feel as if *Q* is intrinsically unpleasant. The next section concerns this mental state.

5.4 UNPLEASANTNESS

This section explores the nature of the unpleasantness of pain and its relationship with motivation.[18] I will provide unitary and composite understandings of unpleasantness, and contingent and necessary understandings of the relationship between unpleasantness and motivation. By the contingent understanding we are motivated by pain because it is unpleasant, and by the necessary understanding pain is unpleasant because we are motivated by it. This complex topic is difficult to settle analytically and there is little or no empirical evidence which can help with this explanatory task. In the end, the decision comes down to competing intuitions. On balance, I favour a composite understanding of unpleasantness and a contingent understanding of motivation. Given the difficulty deciding this matter, it is fortunate that both positions are consistent with the rest of my thesis.

This is because these would be ontogenetic functions of some tokens of *Q*. I discuss the crucial distinction between ontogenetic functions and primary and secondary functions in section *5.1.1*, this chapter.

[18] Consideration of Austen Clark's argument that 'painfulness is not a quale' (2005) has provided much of the inspiration for this section even though it only features explicitly on occasions.

In this section, the distinction I make between a 'motivational mental state' and 'being motivated' is important. A *motivational mental state* is a mental state that has a motivational function. My view is that pain is a motivational mental state because it has the function of motivating injury-preventing behaviour. The claim that a mental state is motivational does not imply that the function is being satisfied; that is it does not imply that it necessarily motivates its subject. By contrast, I am taking mental states like aversion, dislike, desire, etc. to be motivating. If a subject is averse she is *being motivated*. An aversion is an example of a motivational mental state that necessarily motivates its subject.

Asymbolia has been a useful resource for understanding the nature of *Q*, but it proves less helpful for gaining insight into unpleasantness. On a superficial consideration of asymbolia it is tempting to conclude that *U* is identical to unpleasantness, that *U* is necessary and sufficient for (the relevant sort of) unpleasantness. If this is right, there is a bi-directional independence between unpleasantness and tokens of *Q* (tokens of thermal and mechanical phenomenal qualities); in principle *Q* and *U* can be experienced independently of one another. But asymbolia could also be explained by the loss of a necessary component of unpleasantness; that *Q* and *U* are jointly necessary and sufficient for both pain and the relevant sort of unpleasantness (the unpleasantness of pain). In which case, the independence between tokens of *Q* and unpleasantness is unilateral; unpleasantness cannot be experienced in the absence of a token of *Q*. On this possibility, asymbolia is explained by either an inability to evoke *U* or an inability to connect *U* and *Q*.

This analysis is complicated by consideration of the intuition that an experience that is not disliked to some degree could not be unpleasant. By this intuition there is a necessary relationship between unpleasantness and a mental state that necessarily motivates. Some may rail against the claim that dislike necessarily motivates, but I do not mean that the subject necessarily acts. All that is required is for dislike, aversion, desire and so on to, using Austen Clark's metaphor, move at least one lever, cog or pulley of our motivational machinery (2005, p.p.185-186). So in the case of pain, unpleasantness and motivation are necessarily related. If an aversion is necessary for unpleasantness, and unpleasantness is necessary for pain, then:[19]

i) Unpleasantness is constituted by aversion (*A*), a further mental state (*X*) and *Q*; where *A* and *X* constitute *U*, and *U* (*A* and *X*) and *Q* are necessary and sufficient for both unpleasantness and pain.

ii) Unpleasantness is constituted by *A* and *X*; where *A* and *X* constitute *U* and *U* (*A* and *X*) are necessary and sufficient for unpleasantness, and *U* and *Q* are necessary and sufficient for pain.

iii) Unpleasantness is constituted by *A* and *Q*; where *A* is identical to *U* and *U* (*A*) and *Q* are necessary and sufficient for both unpleasantness and pain.

iv) Unpleasantness is constituted by *A*; where *A* is identical to *U* and *U* (*A*) is necessary and sufficient for unpleasantness, and *U* and *Q* are necessary and sufficient for pain.

[19] I am following Clark by referring to 'aversion'. But like Clark I am taking 'aversion' to be a placeholder (2005) for negative desire-like mental states. For the purpose of my thesis, it does not matter whether motivation is cashed out in terms of dislike, desires or any other similar mental state.

I have introduced *X* to avoid identifying *U* with an aversive mental state, but it is clear from (*i*)-(*iv*) that *X* does not offer any explanatory advantages. Indeed, *X* would be an additional explanatory obstacle. It is of course possible that *U* involves another mental state, but then *U* could involve any number of mental states. For these reasons I reject (*i*) and (*ii*). I am assuming that aversion is a transitive mental state; that is a subject cannot be averse without being averse to something (for example, a token thermal phenomenal quality – a token of *Q*).[20] As the aversion is independent of *Q* by (*iv*) I also reject this understanding of unpleasantness and pain. This leaves (*iii*) as the best understanding of the claim that aversion is necessary for unpleasantness. By this understanding both unpleasantness and pain are constituted by a token thermal or mechanical phenomenal quality (*Q*) and an aversion directed at that token quality (*U* – a *Q*-directed aversion). One consequence of the transitivity of *U* (aversion) would be that *U* is not a phenomenal quality. If it were a phenomenal quality, it would be independent of other mental states.[21] On this understanding of the position that unpleasantness necessarily motivates, asymbolia is explained by either an inability to evoke *U* (a loss of the ability to be averse) or an inability to connect *U* and *Q* (a loss of the ability to direct an aversion at *Q*).

In summary, two contingent and one necessary understanding(s) of the relationship between unpleasantness and motivation are on the table. The main differences between these three understandings are:

[20] I am taking motivational mental states like aversion, dislike and so on to be transitive because it seems to me that a subject must be averse to something. If this were not the case Clark's motivational machinery would be moving without purpose; a subject would be motivated by an aversion without being averse to anything.

[21] On a standard reading, phenomenal qualities are irreducible mental states (see chapter 1, section *1.2.1*, fn.12). In essence, this is Clark's argument that painfulness is not a quale (2005); see below, this section.

M1) *U* is a motivational mental state that is necessary and sufficient for unpleasantness; i.e. *Q* (token thermal or mechanical phenomenal qualities) and *U* (unpleasantness) are bilaterally independent. *U* is not sufficient for the right kind of motivation; [22] *U* and an aversion are not sufficient for motivation. *Q*, *U* and an aversion are necessary and sufficient for motivation.

M2) *U* is a motivational mental state that is necessary but not sufficient for unpleasantness. *Q* and *U* are necessary and sufficient for unpleasantness; i.e. unpleasantness is dependent on *Q*. Unpleasantness (*Q* and *U*) is not sufficient for motivation. *Q*, *U* and an aversion are necessary and sufficient for motivation.

M3) *U* is an aversion that is necessary but not sufficient for unpleasantness. *Q* and *U* are necessary and sufficient for unpleasantness; i.e. unpleasantness is dependent on *Q*. Unpleasantness (*Q* and *U*) is sufficient for motivation.

M1, *M2* and *M3* are intended to highlight the differences between three competing positions. In contradiction of the intuition that we are necessarily motivated by unpleasantness, both *M1* and *M2* present contingent understandings of the relationship between unpleasantness and being motivated (an aversion); it is possible to experience *Q* as if it were intrinsically unpleasant without moving a single part of the motivational machinery. According to *M3*, pain necessarily motivates. Additionally,

[22] Henceforth, in *M1*, *M2* and *M3* 'motivation' refers to 'the right kind of motivation' – the motivation of injury-preventing behaviours or responses.

M3 is the only characterisation in which the nature of *U* is specified; *U* is constituted by a *Q*-directed aversion. By *M1*, unpleasantness is an irreducible mental state that in principle can be experienced independently of tokens of *Q* and the subject being motivated (*Q*-directed aversion). By *M2*, the right sort of unpleasantness cannot be experienced independently of tokens of *Q*, but unpleasantness is independent of motivation (*Q*-directed aversion). By *M3*, the right sort of unpleasantness cannot be experienced independently of either tokens of *Q* or motivation (*Q*-directed aversion). In all cases, *Q* is constituted by tokens of affectively neutral thermal and mechanical phenomenal qualities, *U* is a motivational mental state which makes it feel as if *Q* is intrinsically unpleasant, an aversion is necessary for the subject to be motivated and *Q* and *U* are necessary and sufficient for pain.

In his 2005, Austen Clark argues that unpleasantness is not a phenomenal quality. In brief:

I Phenomenal qualities are non-relational.

II The unpleasantness of pain is relational because it necessarily motivates.

III The unpleasantness of pain is not a phenomenal quality.

In effect his argument for the crucial premise (II) amounts to an appeal to the intuition I mentioned above, that if something is unpleasant we are averse to it, where to be averse is to be motivated. But for all that has been discussed, this is just an intuition,

so it is a viable possibility that unpleasantness is constituted by an independent mental state (this is *M1*) that could be a phenomenal quality in the strong sense of colour experience. Here are two reasons for rejecting the view that unpleasantness is a phenomenal quality in this strong sense.

The first, which I call '*dependence*', is that there is a dearth of evidence supporting the independence of (the relevant sort of) unpleasantness. If it were possible to experience unpleasantness in the absence of *Q*, there should be evidence of this phenomenon. The only evidence I am aware of is a *single* case study and this should be treated with caution. In particular, the experimental design and conclusions concern the 'lateral pain system'[23] which is linked with what the subject appeared to lack (*Q*) rather than what seems to have been retained, namely a vague poorly localised unpleasantness (Ploner et al, 1999).[24] Even if this case is taken at face value another explanation is possible. The precise extent of loss of sensation is not clear from Ploner's account, though he is clear that vibration sensation has been retained. So this subject may have retained the slight mechanical sensations associated with deep structures like joints, so phenomenal qualities could be constituents of this subject's unpleasant experiences (i.e. the subject is experiencing *U* that is targeted at slight, poorly localised mechanical sensations). But whether or not this explanation is right, I am not aware of any unambiguous evidence of the independence of unpleasantness.

[23] I outline the lateral pain system in chapter 3, section *3.2.1*.
[24] Nikolas Grahek uses this case as evidence for his position that the affectively neutral phenomenal qualities of pain and unpleasantness are bilaterally dissociable (Grahek, 2007, pp95-140).

The second, which I call '*recognition*', concerns someone with asymbolia's identification of experiences of *Q* as pain in the absence of unpleasantness. If unpleasantness (*U* in *M1*) is a phenomenal quality then a pain is constituted by the phenomenal qualities *Q* and *U*. So it is reasonable to assume that an experience constituted by *Q* and *U* would feel significantly different from an experience constituted by *Q* alone. I have argued in chapter 1 section *1.2.2*, that those with asymbolia utilise the same recognitional concept (*REC*) as normal subjects when they identify their experiences as pain. How then is someone with asymbolia's ability to identify *Q* as pain to be explained?

If unpleasantness is a phenomenal quality in the full-blown sense, it would expected that the post-morbid (*after* brain damage in the case of asymbolia) recognitional abilities of those with asymbolia would be severely compromised. Consequently, they would not be able to identify most of the relevant experiences of *Q* (i.e. those that would have been experienced in conjunction with *U* pre-morbidly) as pain under *REC*. As the evidence is only that they identify *some* of these experiences of *Q* as pain, the claim that unpleasantness is a phenomenal quality is consistent with the evidence. Despite this consistency, I do not find this explanation persuasive. Which tokens of *Q* might someone with asymbolia identify as pain? It seems that the only likely candidates would be those where the contribution of *U*, pre-morbidly, would have been slight. If the contribution of *U* would have been slight it is likely that the experience of the token of *Q* would also be of relatively low intensity. The reasoning behind this is that more intense tokens of *Q* are most likely to be causally linked to higher intensities of energy. These intensities are potentially more harmful and hence

intense tokens of *Q* would in most circumstances (i.e. where background 'modulatory factors' are not a significant influence) be linked to higher intensities of *U*. This reasoning is supported by the medial and lateral pain systems of science. From the first synapse in the spinal cord sensory input diverges into parallel pathways and passes to the brain areas associated with *U* and *Q*. *Ceteris paribus*, the intensity of the input that reaches each brain area is similar.[25] So in the absence of *U*, high intensity tokens of *Q* are unlikely to be recognised as pain because the contribution of the phenomenal quality *U* to the composite phenomenology of pain would be considerable. But then lower intensities of *Q* that would have been experienced as a composite with *U* pre-morbidly would, in the absence of *U*, be difficult to distinguish from any other thermal and mechanical sensations (any other tokens of *Q*). I do not think that someone claiming that unpleasantness is a full-blown phenomenal quality has the resources to settle this problem satisfactorily.

The position that unpleasantness has phenomenology in the weaker sense that there is something it is like to experience unpleasantness is more promising. By this I mean that there is a difference for the subject between an unpleasant experience and either an affectively neutral or a pleasant experience. The explanatory problem (*Stretch*) I presented in the preceding section is an example of this difference; there is a difference for the subject pre- and post-1. This difference, so the argument goes, is explained by the phenomenology of unpleasantness.

[25] The *ceteris paribus* clause accounts for modulatory factors. (For an account of the medial and lateral pain systems see chapter 3, section *3.2.1*)

This contrast certainly highlights a difference in what it is like for the subject. I have no objection to these differences being described in terms of phenomenology. In this sense we might say that there is a quality (or phenomenology) of unpleasantness but this is not enough for the claim that unpleasantness has a subjective character that is *distinct from* (in this case) *Q* rather than an attitude towards *Q* as Clark (2005) would have it, or an adverbial qualification of *Q* as Aydede (2014) suggests. These latter views are inconsistent with *M1*. Although *dependence* counts against the view that *U* can be experienced independently of other phenomenology, I do not think it is strong enough to rule out a modified version of *M1* in which *U* is a phenomenal experience in the weak sense. So *M1* remains a live possibility.

By contrast with *M1* (and *M2*), *M3* is appealing because it is parsimonious, just two mental states, *Q* and an aversion, are required for motivation. It also taps into the intuition that the unpleasantness of pain cannot fail to arouse an aversion. However, *M3* is a problem because of the necessary relationship between unpleasantness and motivation. As unpleasantness (and pain) is constituted by a phenomenal quality (a token of either thermal or mechanical phenomenal qualities) and a motivation (*Q*-directed aversion), there is no need for the subject *to be averse to* the unpleasantness of pain to get motivation. Therefore, unpleasantness has no functional role in motivation. This conclusion cannot be avoided by ascribing higher-order functions to unpleasantness because these motivations would fall under *M2*. For example, the unpleasantness of pain motivates learning. But as there is no necessary relationship between unpleasantness and learning, it is an example of *M2* motivation. Everyday cases like *Stretch* provide *prima facie* reasons to reject *M3*. The unpleasantness of

pain is motivationally relevant; we are averse to pain because it is unpleasant, it is not that pain is unpleasant because we are averse to it.[26]

None of this would matter if *M2* were clearly the better option. *M2* is consistent with our intuition about cases like *Stretch*, but it too faces a significant problem. *Q* and a motivational mental state (*U*) are necessary and sufficient for *Q* to be experienced as if it were unpleasant, they are not sufficient for motivation. So in principle we can experience unpleasantness independently of aversion or any other motivation (in contradiction of Clark's second premise).[27] It is very difficult to imagine ourselves experiencing pain and failing to be motivated to some degree.

The problem here is one of two compelling but competing intuitions; first, that we are motivated by unpleasantness, and second that unpleasantness necessarily motivates. Minor tweaking cannot solve this conundrum. It would not matter if desire, dislike etc. were to be substituted for aversion. The intuitions hold even if 'aversion' is taken to be a placeholder for whatever species of motivation is explanatorily most satisfactory. Similarly, it makes no difference if the "other" *relatum* of unpleasantness is not a token of *Q* or if, contra the argument above, unpleasantness is a phenomenal quality. The most obvious solution to this problem is that one of these intuitions is misleading. As the position that the unpleasantness of pain is causally ineffective is implausible the most obvious candidate is the intuition that underpins *M3*.

[26] David Bain considers this point in much more detail in his 2012 (particularly pp.S80-S81).
[27] Asymbolia is not an actual example. Those with asymbolia explicitly report that their pains are *not unpleasant*.

If asked whether we can imagine experiencing pain without *any* aversion or dislike most of us would, I suggest, reflect on the affect of the unpleasantness of pain; "I am always averse to my pains because they are unpleasant". This is not a constitutive claim like *M3*. Instead, it is a relation between unpleasantness and aversion. A relation like this cannot be necessary. It is possible that desires, dislikes, preferences and the like can change. Clark acknowledges this but does not seem to think it matters so long as the machinery of motivation is moved to some degree. The problem is that an aversion to *Q* is the opposite of a desire for *Q*, as the former is constitutive of unpleasantness by *M3* the latter is constitutive of pleasure. How then is a desire for unpleasantness to be explained?

By *M3*, an aversion and *Q* constitute the unpleasantness of pain. In certain contexts masochists have a desire for this unpleasantness so they have a desire which is directed at an unpleasant experience (pain) constituted by an aversion and an affectively neutral phenomenal quality. The positive desire does not replace the aversion. If it did, then the desired unpleasant quality of pain would disappear (to be replaced by a pleasant experience I presume). If the constitution of unpleasantness is to be partly conceived in terms of mental states these must be negative mental states like aversion, dislike or a desire to avoid. In the case of masochism these negative mental states arouse a positive mental state a like or a positive desire.

Masochism is perfect example of the contingency of the relationship between unpleasantness and particular motivations. As unpleasantness does not necessarily arouse an aversion, or a dislike, or a desire to perpetuate or cease, it is reasonable to

assume that unpleasantness does not necessarily arouse any motivation. Indeed, we might point to very slight pains that we can easily ignore or fail to notice from time to time in support of the claim that unpleasantness does not necessarily motivate. If aversion necessarily motivates then aversion is not a *constituent* of unpleasantness.

In principle, I am not averse to identifying *U* with aversion (pun intended), but it is confusing when considered in conjunction with our attitudes towards the unpleasantness of pain. In everyday thought we are averse to the unpleasantness of pain it is not that we think that pain is unpleasant because we are averse to it. The use of 'aversion' in the constitutional sense does not reflect our commonsense use of the word, nor does it reflect what is going on in masochism cases. Usually we are averse to unpleasant experiences but masochism is evidence that this is a contingent relation.

Because understanding could be compromised by these different levels of aversion, I will proceed by re-adopting the placeholder *U* – by *M3* the unpleasantness of pain is constituted by *Q* and *U*, and *Q* and *U* are sufficient for motivation. So the question is whether the positive desire for pain (for the composite, *Q* and *U*) experienced by a subject in a masochistic context is at the level of mental states that are necessarily motivating or whether *U* is at the motivating level. Masochism is evidence that the mental state(s) that constitute the unpleasantness of pain can be dissociated from either negative (like aversion) or positive desires so there is no obvious reason to deny that sometimes they trigger no mental state at this level. On my understanding of his metaphor this means that no parts of the motivational machinery are moving. However, it might be that Clark's motivational machinery is not limited to the subject

being motivated. Instead he might mean that as *U* has a motivational function the evocation of *U* implies that motivational machinery is moving. If this what he means, I am in complete agreement. On my account, *U* is evoked and targeted at tokens of *Q* by *FS*. As such *FS* might be construed as motivational machinery. But this amounts to a functional claim. This function does not entail that a subject *is* motivated in any way. And so *M3* should be dropped.

What then is the nature of *U* and what makes it a motivation? We might conceive *U* in terms of an adverbial modification – *Q* is negatively affectively toned (Aydede, 2014), or we might persist with terms like 'aversion'. Both terms face a *heterogeneity problem*.[28] This problem boils down to the question of whether or not there is a single species of unpleasantness. The unpleasantness of the taste of an olive, the sound of fingernails being scraped down a chalkboard or the sight of blood might all be described in terms of aversion or negative affective tone but, so the objection goes, these terms fail to capture the distinct unpleasantness of each experience.

It might be responded that the apparent difference between the unpleasantness of pain and these other experiences is given by the *intensity* of the affective tone (say) or the differences between the phenomenal characters of the other constituent of the respective experiences (i.e. the difference between mechanical and visual phenomenal qualities rather than differences between the nature of the negative affective tone). Although the heterogeneity problem is interesting, for my account it does not matter whether there is just one or many 'negative affective tone(s)' or 'aversion(s)'. My

[28] Aydede (2014) uses this term.

objection to claims that one characterisation is better than another is that none really capture what it is like to experience the unpleasantness of receiving dental treatment without an anaesthetic. 'Aversion' fails to capture experiences like this because its use in this context is at odds with everyday usage; we do not normally think of aversion as a constituent of unpleasantness. 'Negative affective (or hedonic) tone' sounds good but I am not convinced it does a better job than 'whatever it is that makes *Q* feel as if it is intrinsically unpleasant and tends to motivate its subject'. But for the sake of brevity 'negative affective tone' is to be preferred over the 'whatever it is…' sentence. So *U* is constituted by 'negative affective tone', which is an affective shading of *Q* derived from its injury-preventing function rather than an adverbial qualification of *Q*. On this understanding the difference between an experience constituted by a *particular* quality and intensity of *Q* (call it *QM**) at a particular bodily location and an experience constituted by *QM** and *U* at the same bodily location, is that the latter is coloured by negative affective tone so that it is experienced as if it were intrinsically unpleasant and tends to motivate its subject.

This account of unpleasantness does not entirely settle matters between *M1* and *M2*, though it seems to me that if there were no *Q* to be shaded by the negative affective tone of *U*, there would be no experience of *U*. For this reason, I favour *M2*. But my thesis is compatible with *M1*. It is even consistent with *M3* even though I reject this necessary understanding of the relationship between unpleasantness and motivation.

In summary of this section, *Q* and *U* are necessary and sufficient for pain and (the relevant sort of) unpleasantness; where tokens of *Q* are constituted by thermal or

mechanical phenomenal qualities and *U* is negative affective tone that makes it feel as if *Q* were intrinsically unpleasant, and an experience of pain is not sufficient for motivation.

5.5 THE EVOLUTIONARY FUNCTION OF PAIN

To this point, I have glossed over the function of pain by describing it as a composite mental state that motivates injury-preventing behaviour. Given the evidence provided by pain asymbolia, peripheral neuropathies and congenital analgaesia it is extremely doubtful that this is not the primary function of pain.[29] However the detail of this crude characterisation is in question because experiences of pain are associated with complex nocifensive behaviours. For example:

i) If we experience ankle pain when walking we tend to limp or sit down.

ii) If we feel an aching ankle when sitting, we might get up and move around.

iii) Alternatively, we might rub or press an aching ankle.

iv) A sudden burning pain may be accompanied by a vocalisation like a yelp or shout.

v) Some pains are causal factors in future behaviour, like taking care when we remove items from the oven in the future.[30]

[29] See chapter 1, sections *1.2.3* and *1.2.4*.
[30] Bain argues that it is difficult for imperativists like Hall and Klein to capture the complexity of pain behaviours in (negative) imperative terms (2011, pp176-179).

While the simple functional characterisation that pain motivates injury-preventing behaviour has been useful, it cannot capture the complexity of these behaviours. On the face of it, the examples expressed in (*i*) and (*ii*) concern the prevention or minimisation of tissue damage; whether walking (*i*) or sitting (*ii*), pain is motivating us to change our behaviour in order to avoid or minimise tissue damage. Likewise, the effect of learning expressed in (*v*) seems to be one of avoiding or minimising future tissue damage by motivating behavioural change. However, an aching ankle (*ii*) may motivate its subject to change position because it would ease joint swelling that is inhibiting recovery. In which case, (*ii*) is an example of the promotion of recovery. This consideration raises the possibility that pain, which is generally conceived in negative terms of motivating subjects *not to behave in certain ways*, may sometimes have a positive function; in some cases of (*ii*), subjects are being motivated *to behave in certain ways*. In a similar vein (*v*) might be viewed positively, pain can motivate subjects to take more care in the future. As such, (*ii*) is open to construal in the terms of recovery. Likewise, tending behaviours (*iii*) like pressing, rubbing and licking are perhaps more about the promotion of recovery than avoidance or minimisation. Vocalising (*iv*) is harder to pin down. On the one hand, it may be a learned response because it brings advantageous attention from others of the same species or social group, in which case it is an example of ontogenetic utility. On the other hand, it could be a secondary (evolved) function. Finally, (*i*)-(*v*) are not exhaustive of the behaviours which are associated with pain.[31]

[31] Complex learned behaviours, like visiting the doctor or taking analgaesics are linked, to pain, but these are clearly not evolved functions. Behaviours like this have exerted no evolutionary pressure on pain so they are not relevant. However, this does not mean that our ability to utilise pain in cognitive processing has not been influenced by evolution.

A fully worked-out version of the evolutionary function of pain is beyond the scope of and is not needed for my thesis. An approximation will serve as a framework to enable me to address P1-P6. The following roughly captures (*i*)-(*v*):

> (*EF*) The ability to experience pain has "evolved to motivate behaviour which avoids or minimises tissue damage, or promotes recovery" (Wright, 2011, p.42).[32]

The phrase 'avoid tissue damage' is intended to convey the prevention of damage to both undamaged tissue and tissue that was damaged on a previous occasion, while 'minimise tissue damage' is intended to apply to the minimisation of the severity of damage that is occurring to either undamaged or damaged tissue. I also want to be clear that it may be that no mental state (whether composite or not) has a primary function as complex as *EF*. But this does not matter so long as *EF* contains a rough characterisation of the primary function of pain and that *EF* excludes what I am calling 'ontogenetic functions'. With respect to the former, it is implausible that *EF* does not roughly characterise the primary function of pain. And with respect to the latter, the inclusion of ontogenetic functions would lose any meaningful sense of what it means to say that pain is motivational, perceptual or both. On top of behaviours (*i*)-(*v*) we also tend to suspect we are damaging tissue when we experience pain and medical practitioners use pain as a means of diagnosing pathology. So humans clearly *utilise* pain in a perceptual role. But this carries no implication that pain *is* perceptual in the sense expressed in section *5.1*. If we were to accept ontogenetic functions then

[32] *EF* is based on the definition of pain I tentatively proposed in a critical account of the IASP's definition of pain. In the final section of this chapter we will see why the definition I proposed – "Pain is the experience that has evolved to motivate…" – is not adequate.

pain is at least motivational, perceptual and introspective (because some of us use pain as a means of considering conscious experience). There is more to the claim that pain *is* a motivational experience (or a mixed perceptual and motivational or a perceptual experience) than mere utility.

The point of this section is that I am claiming that pain fulfils the functional role expressed in *EF*. A perceptualist faces the considerable challenge of expressing this function in perceptual terms. In chapter 2, section *2.1.4* I argued that perceptualism does not have sufficient resources to meet this challenge. Both mixed theorists and motivationalists explain pain in motivational terms. The difference between these theories is that the former are obliged to explain the role that the perceptual content of *Q* fulfils in *EF*, while the latter must explain *Q* and *U* in motivational terms that are consistent with *EF*. Even if a consistent account could formulated, and I will argue that it cannot, I have argued in chapter 2 section *2.3* that the mixed theory cannot address the problems posed by P4 and P6. By contrast, I will provide a motivational account of *Q* that is consistent with *EF* and has the power to explain all of P1-P6.

5.6 THE NOCIFENSIVE FUNCTIONAL SYSTEM

Pain functions as a means of motivating nocifensive behaviours and responses.[33] As such it is an *output* of a system (or systems) that function for the purpose of nocifense (*FS*). A functional system is an *explanatory framework* that is limited by function. If a physiological process in an anatomical structure, a conscious experience, response or

[33] See *EF* in the preceding section.

behaviour is functioning for nocifensive purposes then it is a constituent of *FS*. I am not making any grandiose claims about *FS*. It is simply a useful construct for my purposes. Indeed, part of the explanatory advantage of *FS* is that it is based on the platitude that organisms exhibit behaviours and responses which prevent or minimise harm and promote recovery. Therefore, organisms have systems that yield these nocifensive behaviours and responses. *FS* is an umbrella framework for these systems. One of its merits is that it is not limited by current understanding of anatomy, physiology, consciousness, responses or behaviours and their relationships. So the question of whether the pain matrix is an accurate way of conceiving the anatomical structures that are involved in pain can be answered by considering it in conjunction with other elements of *FS*. *FS* yields predictions. For example, if noxious intensities of energy are not discriminated peripherally then they must be discriminated by CNS structures.[34] The importance of *FS* for my thesis is that its combination of features allows me to make useful generalisations that are not limited by *my* knowledge of anatomy, physiology or function. For example, the statement that mechanically sensitive primary afferent neurons provide input to *FS* (they provide sensory input that is utilised for nocifensive purposes) is uncontroversial and it is useful. It enables me to build a functional account of the systems involved in pain without making controversial claims about the relevant anatomy and physiology.

In essence, the challenge posed in this thesis is that a viable conception of pain, that is our understanding of a potential output of *FS*, must have the power to explain P1 to P6 *and* it must be consistent with other elements of *FS*. I have argued that current

[34] The discussion of this particular aspect of *FS* is the focus of this section.

theories do not have the necessary explanatory power. The most important aspect of my thesis concerns the uncontroversial observation that *FS* involves the discrimination of noxious intensities of energy. Analysis of the concept of noxious intensities of energy shows that the task of discriminating such energy is beyond the capacities of peripheral mechanisms, so the concept of a nociceptive system is inconsistent with other elements of *FS*. Some of the consequences of this conclusion have been discussed in the preceding sections of this chapter. In summary, the view that pain is constituted by a phenomenal quality which is specific to pain and a further mental state that makes it feel as if that quality were intrinsically unpleasant and that pains are not necessarily unpleasant is unsustainable. Instead the phenomenal qualities that are constituents of pain also constitute innocuous thermal and mechanical sensations. There are constitutional and functional differences between these innocuous sensations and pain. A token of the former is an irreducible experience constituted by a token phenomenal quality from the quality space that subserves either thermal or mechanical sensation (i.e. it is constituted by a token of *Q*). Pain is a composite experience constituted by a token of *Q* and a further mental state (*U*) that makes it feel as if *Q* it were intrinsically unpleasant; i.e. *Q* is shaded by negative affective tone provided by *U*. Pain cannot have the same function as innocuous sensations because the former does while the latter do not motivate subjects in accord with *EF*. However, this does not imply that *Q* fulfils different functional roles in innocuous sensation and pain. It might be that tokens of *Q* fulfil a perceptual function in both types of experience.

5.6.1 The argument against the mixed theory

I have characterised the function of pain in terms of behaviour, but the following is also consistent with my arguments:

> (*EF′*) The ability to experience pain has evolved to motivate behaviour which reduces or avoids noxious intensities of thermal and mechanical energy[35]

The difference between *EF′* and *EF* is that the former makes explicit the role of energy in tissue damage, while it is implicit in the latter. We avoid or minimise tissue damage by behaving in ways that moderate the demand imposed by energy. The emphasis in *EF′* suggests perception; we are motivated to moderate the energy represented by *Q*. This understanding is consistent with my analysis of the nature of the affectively neutral component of pain. All tokens of *Q* are perceptual states that represent an intensity of thermal or mechanical energy at a bodily location. When *FS* attaches *U* to *Q* it is in effect making an intensity of thermal or mechanical energy at a bodily location the target of an aversion. Hence, we are being motivated by the *content* of *Q*. This is the mixed theory; *Q* is perceptual and *U* is a motivational because the latter functions as the means of focusing the capacity for aversion, which is necessary for a subject to be motivated, at the energy represented by *Q*.

[35] The element (of *EF*) of promoting recovery is less easily translated. For the sake of argument this problem can be ignored.

Although coherent, the claim that the perceptual content of *Q* fulfils a functional role in pain is questionable. The claim that all tokens of *Q* are perceptual amounts to the claim that all tokens of *Q* have accuracy conditions. Given the influence of modulation, there are serious doubts about the accuracy of the tokens of *Q* that partly constitute pain; i.e. the evidence is that the intensity of *Q* fails to accurately represent the intensity of thermal and mechanical energy.[36] But the potential for this inaccuracy does not seem to matter for the purpose of *EF′*. It does not seem to matter if the intensity of a token of *Q* accurately represents the intensity of thermal and mechanical energy. What matters from a functional perspective is that the intensity of the noxious threat (the intensity by which the energy exceeds the noxious threshold) is *at least* matched by the motivational force of the unpleasantness; i.e. the force of the negative affective tone contributed by *U*.[37] So the *accuracy* of the intensity-representing content of *Q* is irrelevant for pain to fulfil the function expressed in *EF′*. Importantly, this argument does not contradict that either the intensity of *Q* fulfils an important functional role in pain or the accuracy of information about the intensity of energy that is coded at a *sub-personal* level is not important for *FS*.

With respect to the former, it is possible that *FS* has the capacity to ramp up the intensity of tokens of *Q* that partly constitute pain. This would increase the contrast between a pain and other experiences without increasing the *immediate* motivational force of *U*, the effect being one of attracting the subject's attention and thereby bringing cognitive resources of appraisal into play; appraisal aimed at achieving the

[36] See chapter 1, section *1.2.5* (this is part of problem P6).
[37] As *Q* is constituted by affectively neutral thermal and mechanical phenomenal qualities that constitute innocuous sensations and partly constitute pain, tokens of *Q* cannot have content that the energy is noxious.

most advantageous outcome for the subject, all things considered. In this way, pain can be utilised in a perceptual role, but this would be an example of ontogenetic utility.[38]

With respect to the latter, coding at a sub-personal level seems important for the discrimination of noxious intensities of energy. If *FS* is unable to discriminate the intensity of a given type of energy, then *FS* does not have the resources to evaluate the relationship between energy and the tissues.[39]

The argument that the accuracy of the perceptual content of *Q* has no bearing on *EF′* also applies to the thermal and mechanical qualities of *Q*. For the purposes of *EF′* it does not matter if a thermal quality represented a mechanical stimulus or *vice versa*. So perceptual content about the type of energy fulfils no functional role in pain and the argument that the affectively neutral component of pain is perceptual collapses.

The problem for motivational accounts of *Q* concerns the felt location of pains. Felt locations seem paradigm examples of (non- conceptual) perceptual content. A pain motivates us to behave in a manner that is consistent with *EF′* because *Q* is representing a bodily location that requires attention. When we experience ankle pain the accuracy of the felt location of the *Q* is functioning as a means of motivating appropriate behaviour, be it avoidance, limping or tending. We would be less likely to

[38] My intention here is to highlight that the intensity of *Q may* fulfil a specific motivational function the attraction of attentional mechanisms. This speculative view is not central to my thesis so I leave it at this point.
[39] It may be that the mechanism of evaluating the threat posed by energy is not quite like this at a mechanistic level. (See chapter 6, from section *6.2* on.)

behave appropriately if *Q* was inaccurate in this respect.[40] It is interesting that the accuracy of the location of energy seems important when the accuracy of the intensity and type are functionally irrelevant. Consideration of the location of *Q* also brings into focus the question of analysing what it means to say the location of *Q* has accuracy in the context of the function of pain.

Standardly, we do not attribute locations to phenomenal qualities. When we experience a thermal phenomenal quality as if it were located in the right hand we attribute the location to the energy (to the heat) or to the source of the energy (the stove). But when we are in pain the focus of our aversion is the pain. Consequently, the question of whether *Q* accurately represents the location of energy has no bearing on our motivation. Behavioural changes are aimed at ridding ourselves of pain. If we have behaved in a way that has eliminated an ankle pain, our motivational drive has been satisfied; we are no longer motivated by our pain. The accuracy of the locational content of *Q* also has no bearing on our motivation because we are not motivated by either energy (*EF'*) or the prevention of tissue damage (*EF*). Pain motivates us to rid ourselves of pain. But crucially, satisfaction of the motivation to rid ourselves of pain does not get us the functional outcomes expressed in *EF'* or *EF*, a reduction in the intensity of energy or the prevention of tissue damage, unless there is a connection between behaviour that moderates pain and behaviour that moderates noxious energy. The causal relationship between the energy and *Q* is the primary explanation for this

[40] With the exception of tending behaviour this might be challenged. Sciatica pain (pain in any or all of the buttock, posterior thigh or leg) is most often causally related to mechanical pressure on the sciatic nerve in the low back. The inaccuracy of the representation of the location of the energy does not prevent subjects from behaving appropriately, i.e. by standing up and moving around. Appropriate behaviour is at least some cases a matter of trial and error. However, I am inclined to accept that the location of pains is an important factor in motivating appropriate behaviour.

connection but the accuracy of content about the location of energy is also functionally significant (though see fn.40). Behaviour that moderates pain is more likely to prevent tissue damage *because* of the causal relationship between energy that has the potential to damage tissue and tokens of *Q*, and *because Q* has perceptual content about the location of energy.

I favour *EF* over *EF′*, because the evolutionary pressure comes from the maintenance of bodily integrity. In this way, organisms are best placed to avoid predation, feed, reproduce and avoid infection. From this perspective *EF′* gets the emphasis wrong. With these arguments in place, I am in a position to set out my version of motivationalism.

5.6.2 Near-motivationalism

FS uses pain as a means of motivating behaviour which avoids or minimises tissue damage, or promotes recovery (this is *EF*). Satisfaction of this function requires the discrimination of energy that has the potential to damage tissue. This is a function of CNS structures of *FS*, which constantly monitor sensory inputs to evaluate whether the tissues are able to cope with the energy they are absorbing. If the evaluation is that a particular tissue is not coping with energy then a further evaluation determines whether pain, some other or no nocifensive response is appropriate response given the circumstances of the subject. If it is determined that pain is an appropriate response, then *FS* attaches negative affective tone (*U*) to the token of *Q* (a token thermal or mechanical phenomenal quality) that is representing the energy that *FS* has evaluated

as noxious. To be clear on this, perceptual content presented by this token of *Q* is a constituent of a perceptual system it is not a constituent of *FS*, even though coded information about energy is being utilised at the *sub-personal* level by both the perceptual system and *FS*. Conscious perceptual content presented by *Q* plays no part in the sub-personal evaluation. The attachment of *U* to *Q* changes the function of *Q* from a perceptual to a motivational experience because the resulting composite experience (pain) has the function of attracting an aversion. The aversion motivates behaviours and responses that are targeted at pain, not energy. It is effective as a means of avoiding or minimising tissue damage, or promoting recovery precisely because *Q* and *U* are causally related to energy,[41] and because *Q* has perceptual content about the location of energy.

Now some may claim that my position could be reinterpreted in terms of perceptualism. All tokens of *Q* represent energy. The attachment of *U* to a token of *Q* changes the subjective quality of the experience so that it carries the additional (evaluative) content that the energy is noxious (or bad for its subject). This additional content carries the motivational force of pain. This version of perceptualism is very close to the modified position I presented towards the end of chapter 2 section *2.1.3*, and I reject it for the same reasons. A mental state has the capacity to motivate because it is a motivational mental state; a mental state does not have the capacity to motivate because it is a conscious representation of a sub-personal evaluation that energy is noxious.[42] Perceptions are directed towards input, motivations are directed

[41] The evidence is that *Q* and *U* are causally related to sensory input that is transmitted in parallel pathways to the brain (see also fn.44 this chapter).
[42] Note the change here. In chapter *2.1.5*, the evaluative content of *U* was that the noxious intensity of energy represented by *Q* is harmful or bad for its subject. In chapter 4, I argued that tokens of *Q*

towards output. On this version of perceptualism *U* is facing in both directions. There are also explanatory problems with P4-P6.[43]

The objection that my position is really a version of the mixed position is of greater concern. A perceptual experience like *Q* is identical to its content. If *Q* is a constituent of pain and we are being motivated by pain then we are being motivated by the content of *Q*. Nothing I say here is likely to sway those who hold entrenched views on this matter of identity even though pain challenges the view that a phenomenal quality just is its perceptual content. My response is one of iteration; my motivationalism is based on the claim that the *accuracy* of the content of *Q* fulfils no functional role in motivating behaviours and responses that are targeted at pain. The causal relationship between pain and energy and *Q*'s content about the location of energy explains how pain-directed behaviour fulfils the nocifensive function expressed in *EF*.[44] This can hardly be described as a perceptual account of *Q*. A perceptualist about *Q* would have it that we are motivated by the content of *Q*. I have argued that this is not the case. Nevertheless, I have to concede that the locational property of *Q* subtly shades my motivational account. The label 'near-motivationalism' is intended to reflect this concession.

Near-motivationalism has the power to explain P1-P6, because it invokes an evaluative process as means of overcoming the problem posed by the need to

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represent intensities of energy, not noxious intensities of energy. Hence, *U* represents that an intensity of energy is noxious here.

[43] See the final few paragraphs of chapter 2, section *2.1.3.*

[44] A causal relation between *Q* and energy does not suggest that *Q* fulfils a perceptual function. There are parallel pathways leading from thermally and mechanically sensitive Aδ- and C-fibres to the brain areas that are associated with both *Q* and *U*, but *U* is not amenable to conception as a perceptual experience. (See the 'medial and lateral pain systems' chapter 3, section *3.2.1*)

discriminate noxious intensities of energy. The evaluation of a tissue's ability to cope with energy is particularly taxing, and if the efficacy of *FS* is judged by the accuracy of this evaluation then it often gets it wrong. Although I lack the resources to provide a detailed account of this evaluative process, with the help of the problems P4-P6, I roughly model some aspects of this process in the final chapter of my thesis.

6 SOLVING THE PROBLEMS OF PAIN

This chapter is devoted to the problems that I presented as a challenge to current theories of pain in chapter 1. I repeat them here:

P1 *Prima facie* pains are constituted by characteristically unpleasant qualities that vary in intensity and location.

P2 Pains have dissociable affectively neutral and affective components.

P3 Those unable to experience pain suffer a greater number of and more significant injuries than normal subjects.

P4 Pains are often experienced in the absence of the stimulus.

P5 The stimulus is often present in the absence of pain.

P6 There is a poor correlation between the intensity of pain and the intensity of the stimulus.

I argued in chapters 2 and 3, that current philosophical and scientific positions cannot adequately explain these problems. In my view, the main reason for these explanatory difficulties is the almost universal assumption that noxious intensities of energy are detected by peripheral mechanisms. In chapter 4, I argued that peripheral mechanisms cannot detect noxious energy and so central nervous system (CNS) mechanisms must fulfil a significant role in the discrimination of noxious energy. Many of the constitutional and functional problems expressed in P1 and P2 simply disappear in the

light of the consequences of this argument. I have dealt with these in chapter 5, but it is worth summarising them here to make clear the explanatory work that is to be done in this chapter.

The asymmetry between the affective phenomenology of pain and the affectively neutral phenomenology of other experiences (P1) is merely an appearance. The typical experiences we identify as pain have a composite structure. As the affectively neutral phenomenal component of this composite lacks proprietary phenomenology it cannot be sufficient for pain so pain *is* a composite experience constituted by affectively neutral thermal or mechanical phenomenology (a token of *Q*) and affective negative tone (*U*) that makes it feel as if *Q* is intrinsically affective.
With this understanding many of the complex constitutional possibilities (I set these out under '*A-pain*', '*C-pain*', *and* '*F-pain*' in chapter 1, section *1.2.3*) that arise from adopting a recognitional understanding that those experiences we identify as pain under *REC* are pains[1] do not arise.

The understanding that pain has this unique constitutional structure suggests that pain can be defined in terms of this structure. But defining pain in this way raises an interesting question about the scope of 'pain'. It is easy to assume that our ability to recognise pains is also explained by their unique constitution; no other experience is constituted by a token thermal or mechanical phenomenal quality and negative affective tone. But this assumption is contradicted by asymbolia. If we generally recognise experiences constituted by *Q* and *U* as pain, why is it that asymbolics

[1] This is '*T*' in chapter 1, section *1.2.3.*

identify experiences of *Q* as pain? I discuss the scope of pain and consider the issue of recognition in section *6.1*.

Although the mismatch between my definition of pain and our recognitional abilities is a potential threat to my position, the explanatory weight of this chapter is devoted to P4-P6. The evidence of a weak correlation between the stimulus and pain is an obstacle that opposing accounts of pain have failed to overcome. The account I provide in section *6.2* concerns crucial details of the evaluative processes conducted by the nocifensive functional system (*FS*). In the subsequent section (*6.3*), I explain problems P4-P6 and develop my account of *FS*, as a means of illustrating how these processes work. In the final section of this chapter and my thesis, I summarise my arguments and position.

6.1 THE DEFINITION OF PAIN

The typical experiences we identify as pain are constituted by an affectively neutral phenomenal quality (a token of *Q*) and an affective mental state (*U*) that makes it feel as if *Q* is intrinsically unpleasant. But the straightforward understanding that pains *are* the experiences that are constituted by *Q* and *U* is challenged by dissociation cases, in which subjects identify tokens of *Q* as pain in the absence of *U*. I have argued that tokens of *Q* are token experiences constituted by the token thermal and mechanical phenomenal qualities that subserve innocuous thermal and mechanical sensations. As these sensations are not pains, an experience constituted by a token of *Q* is not

sufficient for pain. An experience constituted by a token of *Q* and *U* is necessary and sufficient for pain. In more detail:

Pain A subject is experiencing pain if and only if her experience is a composite constituted by a token thermal or mechanical phenomenal quality and negative affective tone.

Although this is a characterisation of the subjective nature of pain, it is not divorced from objectivity. Both the phenomenal and affective components are linked to function. The subjective qualities of a token of *Q* are derived from *Q*'s primary perceptual function as a representation of thermal or mechanical energy. My position that *Q* fulfils a motivational function as a constituent of pain does not affect these qualities. Negative affective tone is an affective shading of *Q* derived from its nocifensive function.[2]

Two inter-related issues arise from this understanding of pain. The first concerns scope and the second recognition. I discuss these matters in the following two sub-sections.

6.1.1 The scope of 'pain'

I have argued elsewhere that the IASP's definition of pain is seriously flawed, but one of the problems with my proposed alternative – pain "is the unpleasant sensation that

[2] A more precise expression of function (*EF*) can be found in chapter 5, section *5.5*.

has evolved to motivate behaviour which avoids or minimizes tissue damage, or promotes recovery" (2011, p.42)[3] – is that its scope appears too broad. Intensely bright visual phenomenology seems to be a pain on this definition. *Pain* is a significant revision which excludes experiences like this, but there does not appear to be a good principled reason to confine the concept of pain to thermal and mechanical phenomenal qualities.

According to *Pain*, an experience constituted by a thermal phenomenal quality (*TQ*) and *U* is a pain, and an experience constituted by a mechanical phenomenal quality (*MQ*) and *U* is a pain. In both cases, the composite experiences (*TQ* and *U*, and *MQ* and *U*) tend to attract an aversion that motivates behaviours and responses aimed at ridding the subject of the composite. This achieves a nocifensive function because of the causal relationship behaviour and *TQ* and *MQ*. As the visual case can be treated in exactly the same way there is no good functional reasons for this restriction.

The reason we restrict 'pain' to composites involving tokens of *Q* seems to be little more than a convention. We typically identify experiences certain experiences as pain. Exploration of these experiences leads to the conclusion that they are constituted by *Q* and *U*, therefore experiences with this structure are pains. To be clear on this I am not saying that all the experiences we could in principle recognise as pain under *REC* are constituted by *Q* and *U* (all pains by *pain*) or that we could in principle recognise all experiences constituted by *Q* and *U* as pain under *REC*.[4] My point is that it would not

[3] I have used this definition as a means of specifying the evolutionary function of pain (*EF*). See chapter 5, section *5.5*.
[4] I give a reason to believe that we could not in principle identify all experiences constituted by *Q* and *U* as pain in sub-section *6.1.2*, this chapter.

be consistent with *REC* if we identified an experience that is partly constituted by a phenomenal quality other than a token of *Q* as a pain. The definition expressed in *pain* honours this convention, but the following may be more scientifically and philosophically acceptable:

Pain' A subject is experiencing pain if and only if her experience is a composite constituted by a token phenomenal quality and negative affective tone that motivates nocifensive behaviours.[5]

By *Pain'*, pain is, just like *Pain*, constituted by phenomenal and affective components so that the phenomenal character of the experience is shaded by negative affective tone, which reflects its nocifensive function. But *Pain'*, unlike *Pain*, is not restricted by convention. It is restricted by its constitution, which in turn reflects function.

There is a good reason to favour each of these versions. On the one hand, the adoption of *Pain'* would represent a significant departure from our commonsense conception of pain. I am not sure whether that departure is warranted because I believe that our commonsense conception has considerable utility.[6] On the other hand, the view that pain is as defined in *Pain* might restrict our theorising and research. In conclusion of this sub-section, I have no intention of committing myself more than to say that the broad definition expressed in *Pain'* is more philosophically and scientifically

[5] The addition of the condition that the experience motivates nocifensive behaviours avoids the problem that smells or sounds might be construed as pains under *Pain'*. (Thanks to Darragh Byrne for pointing this out to me.)
[6] Resnick (2000) agrees while Gustafson (2000) take the opposing view.

satisfactory. Despite this, my use of 'pain' in what remains of this chapter and the conclusion is a reference to pain as defined by *Pain*.

6.1.2 The recognition of pain

My argument that pain does not have proprietary phenomenology in chapter 5 section *5.2*, was an argument that the phenomenology of *Q* is not specifically for pain; tokens of *Q* subserve both pain that has a motivational function and innocuous thermal and mechanical sensations that have a perceptual function. However, negatively affectively toned mechanical thermal and mechanical phenomenal qualities (the composite of *Q* and *U*) are unique to pain. This is a good explanation for our recognitional abilities; we are able to recognise pains because they have a unique compositional structure. But this explanation is contradicted by subjects who identify tokens of *Q* as pain in the absence of *U*, and by plausible examples of experiences that are pains but are not usually identified as such by their subjects.

Subjects with asymbolia, those who have taken morphine, and perhaps some lobotomised subjects identify experiences that are exhausted by *Q*, as pain. These experiences are obviously not identified as such because by they have a unique compositional structure. We have good reason to accept that these subjects are deploying the concept pain in a normal manner so it is reasonable to conclude that the rest of us would make similar identifications if we were taking morphine or had specific brain damage. This strongly suggests that negative affective tone adds the property of unpleasantness to tokens of *Q* without obscuring their phenomenal

character.[7] Plausibly, these experiences are identified as pain rather than innocuous thermal or mechanical sensations because many tokens of *Q* are most often experienced as constituents of pain; i.e. these tokens of *Q* exemplify the phenomenal qualities we associate with pain.[8]

Other plausible cases of misidentification are relevant to the explanation of P5 (in section *6.3.2* below). The IASP write, "Each individual learns the application of the word ['pain'] through experiences related to injury in early life" (IASP 2014). Although this quotation is not explicit, I take it that the IASP are referring to a process of triangulation where words like 'pain', 'hurt' and other synonyms are used when a child exhibits pain behaviour and crucially at moments of potential traumatic injury like falls and hard knocks. Hence, we learn to associate 'pain' with the thermal and mechanical sensations that accompany episodes like this. A little self-reflection reveals that we continue to identify these experiences as 'pain' once we are conceptually mature. For example, if you unexpectedly bang an elbow hard against a wall knock you might well use a pain-related expletive like "Ow!" and think "That hurt". And if questioned you may well assert that the experience was unpleasant. But the force of the assertion will probably have been bolstered by an ache that is often experienced a few seconds after the initial episode. This 'second pain' should not be confused with sudden initial mechanical sensation which I maintain, will often be

[7] This supports the position that *U* is not a phenomenal quality. (See the arguments in chapter 5, section *5.4.*)
[8] Some sudden very high intensity mechanical phenomenal qualities associated with severe injury are not experienced as unpleasant and are not identified as pain so the tokens of *Q* that are identified as pain are not like these. (See below, this sub-section)

affectively neutral.[9] To add weight to my flimsy introspective resources, cases like the Reagan shooting[10] (and perhaps some cases of sudden mechanical injury in sport and war) are evidence that some of the everyday experiences we identify as pain are not unpleasant. These experiences are constituted solely by tokens of *Q* that are identified as pain because of the circumstances in which they occur.[11] This makes functional sense, because episodes like these are so sudden that pain would almost certainly make no difference to the outcome. So we have reason to believe that normal subjects, as well as those on morphine, asymbolics and some lobotomised subjects identify experiences of *Q* as pain under *REC*.

It is also possible that we tend to deny that certain experiences are pain even though they are constituted by *Q* and *U*. My experience is that intensely unpleasant experiences associated with compression (crushing) forces that are spread over a fairly large body area do not seem to have the character of pain. The likely reason for this is it is unusual to experience high intensity but diffuse mechanical phenomenal qualities. We simply have not learned to associate 'pain' with these experiences, even though they are constitutionally and functionally identical to those pains we consider to be paradigmatic.

[9] This is so-called 'second pain', which is related to C-fibre sensitisation. (See Craig 2002, p.664, for a definition of 'second pain'; see also Price, et al, 2002.)
[10] See chapter 1, section *1.2.5*.
[11] It is not because they are high intensity mechanical sensations that would normally be experienced as unpleasant because my contention is that these particular experiences are not often experienced as unpleasant.

Although much of the explanation in this sub-section is speculative, it is no less plausible for that.[12] It certainly provides an explanation for the mismatch between our recognitional abilities and the occurrence of pain as I define it. *REC* embraces a motley collection of experiences that are united by neither their phenomenal character nor their function. We could in principle identify only a proportion (the majority?) of all pains and we misidentify a (significant?) proportion of affectively neutral thermal and mechanical sensations as pain by reference to our memories of the experiences we have learned to categorise together as pain.

6.2 INPUT AND OUTPUT EVALUATION BY FS

Pain scientists conceive pain in terms of three dimensions, one of which, the cognitive-evaluative, refers explicitly to evaluation. In chapter 3 section *3.1*, I expressed my view that this multi-dimensional model is unclear, but I doubt that scientists would object to my conceiving modulatory mechanisms like the opioid system and the influences on these systems like cognition and memory as constituents of an evaluative process. In support of this assumption the eminent scientist Patrick Wall has written, "Could it not be that the sensory input [associated with pain] is analyzed, classified, and defined in terms of what the organism might do about the stimulus?" (Wall 1996, p.125). So the claim that the nocifensive system (*FS*) is partly constituted by a process that evaluates whether, which and to what degree a nocifensive response is appropriate given the nature of the specific threat and other broader considerations that reflect context is not controversial.

[12] These claims are empirically testable. What would be required would be for a large number of subjects to introspect and record their experiences.

By contrast, my claim that the discrimination of noxious intensities of energy is also an evaluative process is new. This claim is based on an argument with the following structure:

I) A nocifensive system requires the ability to discriminate between noxious and innocuous intensities of energy.

II) The fact that pain motivates injury-preventing behaviour is powerful evidence of the existence of a nocifensive system.

III) Humans experience pain, therefore there is powerful evidence that humans have the ability to discriminate between noxious and innocuous intensities of energy.

IV) Noxious intensities of energy cannot be discriminated by relatively simple peripheral nervous system mechanisms.

V) Given III and IV, there is powerful evidence that humans have a relatively complex mechanism involving the central nervous system and peripheral sensory structures that functions to discriminate noxious intensities of energy.

Premises I and II are self-evident and so the lemma (III) is not controversial. I have argued for premise IV in chapter 4. The conclusion (V) simply adds legitimate detail; if noxious energy is not discriminated peripherally, then the CNS must be involved. Some of the difficult explanatory problems I have presented, in particular P6, provide evidence supporting this conclusion. In sub-section *6.2.2*, I present sketches of the input and output evaluative processes that underpin the experience of pain. These

sketches are the basic material that enables me to explain P4-P6. In general, the expectation that pain should correlate with a stimulus is derived from the mistaken view that pain is at least partly perceptual. The idea that there is a relationship between a stimulus and pain is difficult to rationalise with my arguments. Pain is not a response to a stimulus. Nevertheless, if we take it that *FS* most often evokes pain at some intensity in response to an evaluation that an intensity of energy is noxious to some degree, the occurrence and intensity of pain can be construed as a means of assessing the accuracy of the evaluative process. In this light the weak correlations expressed in P4-P6 can be put down to the inaccuracy of the evaluative process. There is more to this "inaccuracy" than the difficulty of determining which intensities of energy are noxious though, the concept of the noxious threat posed by a stimulus cannot be clearly determined.

6.2.1 Noxious intensities of energy

In chapter 4 (particularly section *4.4*), I wrote of the difficulty of discriminating noxious from innocuous intensities of energy when damage thresholds vary because of adaptation or tissue damage. This difficulty is compounded by the fact that information about energy is limited to the stimulus; i.e. the energy absorbed by a receptor. Crucially this energy is distinct from the energy source. Consideration of this distinction significantly complicates the construct of a noxious intensity of energy and provides insight into the functioning of the input evaluating process.

Thus far I have taken a 'noxious intensity of energy' to be an intensity that is at or exceeds the threshold at which energy passes from being innocuous to posing a threat to the tissues. This threshold – the 'noxious threshold'[13] – is distinct from the 'damage threshold' – the threshold at which energy begins to damage. So all damaging intensities of energy are noxious, but only a proportion of noxious intensities of energy are damaging. This distinction reflects the IASP's distinction between a stimulus that threatens tissue damage and a stimulus that is damaging tissue.[14] Although this distinction is a useful construct it obscures details that are of importance to the account of evaluative processes I present in the following sub-section.

The thermal damage threshold – the threshold at which tissue begins to damage – is 47^{o}C so thermal energy at 46^{o}C cannot damage tissue. Why then is thermal energy at 46^{o}C considered to be 'noxious' or 'threatening'? The answer to this question does not concern the intensity of the energy in the tissues because thermal energy at 46^{o}C, cannot damage the tissues. It concerns instead the relationship between the intensity of energy in the tissues and the intensity of the energy source. An energy source with the capacity to elevate tissue temperatures to 46^{o}C is likely to have the capacity to elevate tissue temperatures over the damage threshold. This notion of likelihood is useful. To fulfil the nocifensive function (*EF*) of avoiding or minimising tissue damage *FS* responds to energy that is *likely* to be causally related to energy sources that have the capacity to elevate the intensity of tissue energy over the damage

[13] I defined the 'noxious threshold' in these terms in chapter 2, section *2.1.2*.
[14] This is a translation of the IASP's definition of a noxious stimulus, "A stimulus that is damaging or threatens damage to normal tissues" (2014). See chapter 4 for an analysis of the concept of a noxious stimulus.

threshold. Unfortunately, the construct of a noxious threshold cannot do justice to likelihood, because likelihood is not determined *solely* by the intensity of energy.

The temporal characteristic of energy that is being absorbed by the body is important. The longer a source of thermal energy at a temperature in excess of the damage threshold is in contact with the tissues the more likely it becomes that tissues will damage. A 'noxious threshold' cannot adequately capture the threat posed by the temporal character of energy. To see this consider that the principle behind *FS* responding to a noxious threshold is that once tissue temperatures reach 42^{o}C (the noxious threshold for the sake of the illustration) *FS* triggers an appropriate response. But this principle does not take into account the effect that the intensity of the *energy source* has upon the time available for response. The *rate* at which tissue temperatures rise is an important factor in evaluating whether a nocifensive response is needed; the faster the rise in tissue temperatures the greater the disparity between tissue temperature and energy source. A thermal energy source at 80^{o}C will raise tissue temperatures very rapidly, cutting available response times between a noxious threshold, the damage threshold and beyond to such an extent that adequate response would be impossible.

Sensory input to the *CNS* is being generated moment by moment. Significant moment by moment changes in the intensity of this input represent a significant disparity between tissue energy and an external energy source. The greater the disparity between the energy source and the damage threshold the more rapidly both impulse frequency and the number of neurons firing increases (this is the rate of increase in the

'afferent barrage') and the faster a response is required. In other words, if there is a dramatic increase in the afferent barrage it is likely that the energy has the capacity to damage tissue even though the intensity of tissue energy is well below a hypothetical noxious threshold. This thesis predicts that *FS* has the capacity to identify such changes because they indicate that an energy source has the capacity to damage tissue.[15]

This principle is true of both thermal and mechanical energy but it is more difficult to apply to the latter, because rapidly, increasing intensities of mechanical energy are a feature of normal activity. Running for the bus requires sudden increases and decreases in energy. Significant nocifensive responses to rapid changes like this would seriously inhibit our ability to interact effectively with our environment. Another reason why we do not seem to respond significantly to most rapid changes in mechanical energy is that a large proportion of all the mechanical energy absorbed by the body is internally generated against resistance. The control of this energy is mechanistic (i.e. it is controlled at a sub-personal level), and crucially it is restricted by our capabilities. We simply cannot generate intensities of mechanical energy that exceed our ability to generate mechanical energy. This is not to say that the generation of energy against resistance cannot cause damage; it is of course a fact that we strain muscles, rupture tendons and so on. The detail of the mechanisms that cause injuries

[15] There are good reasons to accept that such changes elicit nocifensive responses. In particular, it explains some of the pain patterns that subjects exhibit in the presence of tissue damage (see section *6.3.3* this chapter). Tendon reflexes are also best explained in these terms. Tapping a tendon with a tendon hammer induces a sudden response in muscles opposing the stretch. This sudden rapidly increasing external force is well below the damage threshold. Although these mechanisms fulfil an important role in movement and balance, they also prevent damage to joints so they can also be nocifensive responses. If reflexes like this were triggered by specific intensities of energy, they would produce inappropriate muscle contraction when we engage in intense activity. So whether a reflex is triggered is best explained as a function of the rapidity of change in the afferent barrage not simply by the intensity of the afferent barrage. The same can be said of thermal energy.

like these is not of relevance here. The point is that *FS* does not seem normally to trigger nocifensive outputs in response to rapidly changing intensities of internally generated mechanical energy, but this does not mean that there are no such outputs as these outputs are likely to be subtle changes in muscle activity to effect posture or movement patterns rather than pain.[16]

Externally generated mechanical energy like knocks and falls,[17] are examples of rapidly increasing intensities of energy and so presumably they are accompanied by rapid changes in the afferent barrage. In section *6.1.2* of this chapter, I gave reasons to think that sudden episodes like this often do not trigger an *immediate* pain because the energy is irresistible,[18] but like internally generated mechanical energy this does not mean that the change in the afferent barrage has not had any effect on *FS* or that *FS* has not produced an output. Rate of change is an important factor in nocifensive responses to mechanical (and thermal) energy in the presence of tissue damage. I will discuss this important issue in the following sub-section.

The other side of the temporal feature of energy concerns *persistence*. Low intensities of mechanical energy have the capacity to damage tissue if they are prolonged. For example, sitting creates pressure which partially occludes circulation to various tissues. This is not a problem in the short term, but sit for long enough and damage could occur. When your buttocks ache, it is a sign that *FS* has evaluated that the temporal characteristics of a more or less fixed intensity of energy represent a threat.

[16] I discussed these matters in chapter 4, section *4.4.2*.
[17] As opposed to external energy that is resisted by muscle action, which is subsumed into the above.
[18] This may seem doubtful. We tend to believe we experience immediate pain when we fall say. I have given reason to believe that this is not always the case. Indeed it may be that we do not experience pain in the majority of cases. I discuss this in more detail in section *6.1.2*, this chapter.

In conclusion, a particular intensity of energy is not a threat unless it is at or above the damage threshold. The relevance (to *FS*) of a stimulus intensity that is below the damage threshold concerns the likelihood that the stimulus is causally related to an energy source with the capacity to cause damage. While intensities of energy that are just below the damage threshold might reasonably be deemed to be noxious and therefore of relevance to *FS*, it would be a mistake to assume that other stimuli are irrelevant. Rapid changes in the afferent barrage and persistent input from mechanically sensitive neurons also represent the likelihood that the stimulus is causally related to an energy source that has the capacity to damage tissue. It is worth adding that the evaluation of temporal aspects of the stimulus cannot be encoded by sensory receptors because it requires relating *moment by moment* information about the stimulus. This is a function of some sort of memory. Given this complexity, my explanation of the evaluation of sensory input by *FS* would not be helped by the concept of a noxious threshold so I will not use it in what follows. A 'noxious intensity of energy' refers to energy that has been absorbed by the tissues and is either damaging or is causally related to an energy source that has the capacity to elevate tissue energy to the damage threshold.

6.2.2 Evaluating sensory input and appropriate nocifensive outputs

In the introduction to this section, I summarised an argument to the effect that humans (at least) have a mechanism for discriminating noxious intensities of energy. This is an evaluative process because noxiousness is not a property of the stimulus; it has to

be evaluated whether or not a stimulus represents a noxious intensity of energy. I also pointed out that it is not controversial to claim that *FS* is partly constituted by a process that evaluates whether, which and to what degree a nocifensive response is appropriate given the context. In short, *FS* is partly constituted by input and output evaluative processes (abbreviated as '*CI*' and '*CO*' respectively) neither of which is limited to CNS activity. In this sub-section, I sketch out details of *CI* and *CO* that will enable me to start explaining P4-P6 (in section *6.3*).

For the purpose of this sketch *CI* and *CO* are best thought of as evaluative processes that function in series; i.e. *CI* discriminates whether input represents noxious energy and passes these discriminations on to *CO*, which decides appropriate nocifensive output(s).[19] In more detail, *CI constantly* monitors input from sensory neurons[20] that are constituents of other functional systems to determine whether and to what degree a stimulus is worthy of a nocifensive response.[21] If it is evaluated that a stimulus is worthy of response *CO* is triggered. *CO constantly* monitors competing pressures on a subject's resources to determine whether, which and to what degree a nocifensive response is appropriate. An illustration of the relationship between these evaluative processes and the functions of the components of pain will be helpful.[22]

[19] This is a little misleading but it is sufficient for modelling purposes here. I develop my account of this relationship in the following section.
[20] The reference to sensory neurons generally rather than thermally and mechanically sensitive neurons is deliberate. It is as an acknowledgement that light and other types of energy can be noxious and elicit nociceptive behaviours. See my discussion of the scope of 'pain' in section *6.1.1* above.
[21] Note it does not determine 'whether and to what degree a stimulus is *noxious*' for all the reasons I have given above; i.e. it is far from clear what 'noxiousness means' if it is considered as a threat rather than a harm.
[22] These are sub-personal *evaluative* processes so they are subject to accuracy conditions. An evaluation (by *CI*) that a stimulus is worthy of response to some degree is accurate if the stimulus *is* worthy of response to that degree in ideal circumstances; i.e. in the absence of other considerations. An evaluation (by *CO*) that a given response is appropriate is accurate if the response *is* appropriate, all things considered. Note this means that a response like pain is the result of an evaluation and could be *interpreted* as a representation of that evaluation. Nevertheless, pain is a motivational mental state.

Thermal energy at 50°C stimulates sensory receptors leading to an increase in the afferent barrage. In response the nocifensive mechanism that monitors sub-personal sensory input (*CI*) evaluates that the stimulus is thermal, has a particular bodily location and that it is worthy of response to some degree so the output side of the evaluation is triggered. *CO*, which is constantly monitoring competing pressures on the subject, evaluates that pain is an appropriate response given the subject's current context and so *FS* attaches *U* to the thermal phenomenal quality that is causally linked to the relevant stimulus (and is an output of a different functional system). Consequently, the subject experiences pain rather than an innocuous (affectively neutral) thermal sensation. This attracts an aversive response that is likely to trump other occurrent motivational states. Consequently the subject attempts to rid herself of the pain, in so doing she removes her hand from the energy source that is causally related to the sensory input that *CI* is evaluating as worthy of response. This example can be generalised by dropping the references to thermal energy so that, "Thermal or mechanical energy stimulates sensory receptors…". This generalised account is the basis for my explanations of P4-P6, but before engaging with these problems I want to address a possible objection that simple reflex responses would be more effective for avoiding or minimising tissue damage.

The advantage of reflexes is that they are significantly quicker than consciously mediated motivations.[23] Indeed, it is likely that this is what would happen if the temperature of the energy source were very high (80°C, say). In such circumstances,

[23] They are quicker because reflexes are simple spinal mechanisms which link sensory input with motor output via a single synapse.

contact would mean that tissue temperatures would rise very rapidly giving rise to a barrage of impulses that occasion reflex withdrawal *prior* to the experience of pain. But if the temperature of the energy source was significantly lower (but in excess of the damage threshold) a subject might pick up a pan full of boiling water only to experience, as she moved towards the sink, a rapidly intensifying pain in the palm of her hand. If a reflex response produced an opening of her hand in such circumstances she would drop the pan and as likely or not scald her legs or other parts of her body. If the relative force of motivational mental states, which are determined by sub-personal functional systems, represented the final word on behaviour, then the subject in this example would have dropped the pan. That she can resist the motivational force of her pain illustrates that pain is a means of fine-tuning behaviour to the advantage of the subject.

With an account of threat (noxiousness) and this introductory outline of the way *CI* and *CO* function together to yield nocifensive outputs I am in a position to begin to explain P4-P6.

6.3 THE WEAK CORRELATION BETWEEN PAIN AND STIMULUS

The problems I presented as an explanatory challenge for philosophers and scientists in chapter 1 section *1.2.5*, have changed significantly in the course of developing my arguments and thesis. In chapter 1, I took the term 'stimulus' to refer to tissue damage in deference to the positions of all the philosophers I have considered for this thesis. In chapter 2, section *2.1.1*, I argued that tissue damage cannot be the stimulus, and

adopted noxious intensities of energy as the stimulus in deference to pain scientists. In chapter 4, I argued that noxious intensities cannot be detected by sensory receptors. The sensory receptors that are of relevance detect thermal or mechanical energy; *CI* discriminates whether the energy is noxious. For this reason, P4-P6 require revision. The problem is that pain is often experienced in the absence of noxious intensities of energy (P4), noxious intensities of energy are present (in the body) in the absence of pain (P5) and pain does not correlate well with the intensity of noxious intensities of energy (P6).

As a consequence of this revision several of the problems I presented under P4 and P5 simply disappear. Stretching a forefinger back into extension, pulling the hair on the back of a hand or sitting on a chair for a long period (P4) are examples in which the occurrence of pain correlates with noxious intensities of energy so they are easily explained by the basic account above. Under P5, I observed that tissue damage is most often present in the absence of pain. This also is no longer a problem; *FS* attaches *U* to tokens of *Q* when *CI* evaluates that the *energy*, not the tissue damage, represented by a pattern of action potentials is worthy of response and *CO* evaluates that pain is an appropriate response. However, second pain, chronic pain like some low back or neck pain, tension headache and trigeminal neuralgia (P4), cases of injurious forces in the absence of pain (P5) and problem cases like '*sprain*' (P6) still require explanation.

6.3.1 Pain in the absence of noxious intensities of energy – P4

Second pain – a dull diffuse ache that often follows a few seconds after a sudden injurious intensity of energy – highlights some of the important features of my account. Second pain is clearly an example of pain in the absence of a noxious intensity of energy because the only relevant energy in the tissues is tissue heat or mechanical pressure influenced by gravity. But it is not just an unhelpful consequence of the chemical environment post-injury. The function of pain is not aimed solely at damage avoiding or minimising behaviours. Pain is also a means of motivating tending behaviours. As *CI* is conceived in terms of an evaluation that energy is worthy of a response rather than an evaluation that energy is noxious it can be said that *CI* evaluates inputs arising from the sensitisation of sensory neurons (including high-threshold 'silent' C-fibres) as worthy of response because tending behaviours promote recovery.

The label 'chronic pain' refers to pain that has persisted for three months or more.[24] Chronic pains can be 'syndromes' like tension headache or trigeminal neuralgia and some low back or neck pain or they can be associated with frank tissue damage like arthritic knees and hips. Tension headache and trigeminal neuralgia are usually explained in terms of pathological changes to sensory mechanisms and 'maladaptive plasticity'.[25] In these cases, the evaluation that energy is worthy of a response is

[24] The word 'persisted' is not intended to convey constant pain. Persistent pain *may* be constant, but much more commonly it is episodic pain at a particular location. For example, knee pain that comes on with weight-bearing but eases with rest.

[25] The ability to change anatomically and physiologically is normally advantageous; hence it is 'adaptive'. In maladaptive cases, the changes are harmful in the sense that pain causes suffering and inhibits activity without benefit. The inhibition of activity promotes further maladaptive plasticity so there is a vicious circle. (See Flor et al, 2006; and Woolf and Salter, 2000.) It is worth noting that cases of maladaptive plasticity can be interpreted as pathological changes.

explained by faulty mechanisms. It is a problem involving *CI* because it concerns either or both faulty input or faulty processing.[26]

Although all chronic low back and neck pains are often lumped together under the 'problem' label, many (probably most) involve unresolved tissue damage. They are a problem because medical practitioners *judge* that these are cases in which pain is experienced in the absence of noxious intensities of energy on the basis of tissue damage. As the connection between the severity of tissue damage and threat is extremely difficult to evaluate there are good reasons to be suspicious of some of these judgements.[27] However, it is undoubtedly the case that many pains experienced by subjects with chronic back or neck pain are not correlated with noxious intensities of energy. The explanation is that *CI* is mistakenly evaluating innocuous intensities of energy to be worthy of a nocifensive response. The reason for the error is explained by the complexity of evaluating which stimuli are worthy of a nocifensive response. Detailed consideration of these cases reveals that the problem concerns the difficulty of evaluating the ability to cope with energy (the ability to supply) – it concerns *CI*. This involves both short- and long-term memory. As I will be discussing the former in my explanation of *sprain*, I will explain chronic low back and neck pain in conjunction with P6.

[26] The distinction between input and processing is useful but misleading because it makes it seem as though input is distinct from processing. In sub-section…I will explain that *CI* and *CO* are not strictly in series. The consequence being that input is a constituent of processing.

[27] Although judgements to the effect that an intensity of energy of energy is or is not noxious may seem to be tangential to my thesis it is of direct relevance. In the main text I claim that the majority of the pains we experience post-injury are most likely inaccurate with respect to some objective notion of what is noxious.

6.3.2 Noxious intensities of energy in the absence of pain – P5

The fact that in circumstances of sport and war subjects often suffer significant injury in the absence of pain is usually given a contextual explanation. In such circumstances the unpleasant affect of pain could distract its subject to such an extent that it compromises survival. So the inhibition of pain is a normal adaptive process in some circumstances. In my terms, *CI* receives input that it evaluates as worthy of a response, but *CO* evaluates that pain is not an appropriate response given the context so *FS* does not attach *U* to *Q*. Consequently, the subject experiences a token of *Q* that is causally related to the high intensity of energy responsible for the damage – a token mechanical quality that is experienced as a thud, snap, etc. – but does not experience pain. As scientists have identified mechanisms like the opioid system that modulate the neurological activity associated with pain, there is an extant mechanistic explanation of *CO*'s ability to turn evaluations that pain is not an appropriate response into actual inhibition (or facilitation).[28]

This straightforward explanation is complicated by the Reagan case.[29] Perhaps his gunshot injuries can be explained, like war injuries, in terms of the evaluation of response even though the context does not seem war-like in any respect. That is, it can be explained in terms of the normal functioning of *CO*. But it is equally amenable to explanation in the same terms as sudden high intensity mechanical stimuli that are often experienced as affectively neutral thuds, snaps, knocks etc., which I discussed in sub-section *6.1.2*, above. That is, it was not possible for Reagan to respond

[28] See Fields (2004) for more on opioid modulation of pain.
[29] See chapter 1, section *1.2.5*.

advantageously to the bullet. The hypothesis is that *CI* does not usually evaluate afferent barrages representing such energy as worthy of response. This raises a doubt about the sport and war cases; does *CO* inhibit pain related neurological activity or does *CI* not evaluate that the relevant input is worthy of response. Although it is clear that subjects who are injured in sport and war do not report second pain, unlike subjects who are suddenly injured in everyday contexts, the conclusion that *CO* does inhibit neurological activity in cases of sport and war is complicated by consideration of the relationship between *CI* and *CO*.

Reports of thuds, snaps and the like are consistent with the view that subjects experience token mechanical phenomenal qualities (tokens of *Q*), in response to sudden high intensity mechanical energy. If this is right, either *CI* is not identifying the stimulus as worthy of a response or *CO* is evaluating that pain is not an appropriate response. Either way, *FS* does not attach *U* to the relevant token of *Q*. In circumstances of sport and war, scientists explain tissue damaging events like these in the absence of pain in terms of context. *CO* has evaluated that pain would not be an appropriate response given the circumstances so *U* is not attached to *Q*. By my sketch (in section *6.2.2*) *CI* and *CO* are in series, this would mean that *CO* must inhibit brain rather than peripheral activity to prevent *U* being attached to *Q*. If it were otherwise the subjects in these examples would experience a pain because the inhibition of peripheral input would be inhibited *after* the evaluation that a stimulus is worthy of response. Assuming strong inhibition, this would prevent pain being experienced subsequent to the damaging event, but central inhibition prior to the damaging event would be required to prevent the initial evaluation by *CI* giving rise to pain.

This may explain what is going on these cases, but it is not a good explanation. With respect to cases of sport and war, the presumption I am working with is that the afferent barrage associated with bullets penetrating tissue and the intensities of energy required to rupture an Achilles tendon is evaluated by *CI* as worthy of a response. This involves the recognition of an impulse pattern. As pattern recognition can be explained in very simple mechanistic terms, it looks as though evaluation by *CI* can be a fast process. By contrast, contextual evaluation (by *CO*) involves diverse inputs that may include perceptual mechanisms, conscious thoughts, emotional states, etc.[30] so it is comparatively slow. Also the effectiveness of the evaluation would not be helped by relatively slow acting peripheral mechanisms. Given that the function of pain is to motivate behaviours which avoid or minimise the effects of energy, a mechanism like this would be too slow to make any difference to the extent of many injuries.

Greater speed can be achieved if the relatively slow process (*CO*) is not strictly in series with the relatively fast process (*CI*). On this account *CO* constantly evaluates context and maintains either the whole of *FS* or the relevant part(s) of *FS* in a state that reflects that evaluation; i.e. an inhibited, facilitated or neutral state.[31] In circumstances like sport and war, *CO* has evaluated and is evaluating that pain would not be advantageous so either there is no afferent barrage because *FS* is in an inhibited

[30] These are the modulatory factors I have mentioned throughout my account.
[31] It is my view that *CO* has the capacity to influence the state of *FS* both generally and selectively. I offer no argument for this here. It is suffice to say that the latter has greater utility, but the former would be very useful in circumstances where all an organisms available resources are channelled towards an important end. Fight or flight comes to mind so war might be a good example of general effect.

state which either involves or does not involve significant inhibition to the peripheral pathway prior to the transmission of action potentials in diverging pathways (i.e. inhibition distal to second order neurons in the spinal cord). As the former would inhibit input to pathways that are necessary for *Q* and the subjects in these examples experience tokens of *Q* the latter is a better explanation of what is going on in sport and war. With regard to *CI*, the difference between circumstances in sport and war, and everyday occurrences is that in the former *CI* is not receiving an afferent barrage and in the latter, it is receiving an afferent barrage. The blurring of the concept that *CI* and *CO* are in series is reflected in the blurring of the content of the evaluations. In the case of sport and war the evaluation is that energy is not worthy of a pain response *because* of the context, and in the everyday context the evaluation is that energy is not worthy of a pain response *because* it is not possible to do anything about the energy.

In summary of this sub-section, cases of noxious intensities of energy in the absence of pain are explained by either *CI* or *CO*. In everyday cases, *CI* evaluates that energy is not worthy of a nocifensive response. In cases of sport and war, *CO* evaluates that pain is not an appropriate response given the context. Consideration of these cases has refined my sketch of the relationship between *CI* and *CO*. *CO* continuously evaluates the context of the subject and sets the state of *FS* accordingly. With respect to circumstances where it can evaluated in advance that pain would not be an appropriate response to noxious intensities of energy, *CO* does not evaluate in response to processing by *CI*. Instead, in these circumstances evaluations by *CO* are prior to evaluations by *CI*. Indeed, *CO* does not require input from *CI*. The recognition of particular impulse patterns by *CI* in effect triggers the attachment of *U* to *Q*. This

account is supported by the existence of descending mechanisms which modulate peripheral input. These mechanisms mediate the effects of cognition, anxiety, and other modulatory factors.

6.3.3 The variable relationship between intensities of energy and pain - P6

The problem of the variable relationship between intensities of energy and pain has been an insurmountable obstacle for the philosophical and scientific accounts I presented in chapters 2 and 3. *Sprain* is a paradigm example of a problem like this:

Sprain A subject with a sprained left ankle has been sitting watching television for a couple of hours. She is pain free even though her ligaments are sprained. She rises from her chair. In so doing, she puts a very small amount of body weight on her left foot and experiences quite intense pain as a consequence. Nevertheless, she carries on and begins to limp around the room. The continued movement is accompanied by a steady decrease in the intensity of her pain and so she slowly increases the intensity of the weight-bearing force on her ankle. After six minutes or so her pain has eased to mild discomfort despite the fact that she is taking most of her body weight on her left foot as she walks. Soon she sits down again, but almost immediately the telephone rings and she stands again. This time her ankle pain is very slight.

The essence of the problem is that the damage threshold of the tissue is constant, while the relevant mechanical energy and the pain are independently variable. If the noxious threshold is taken to represent a safety margin at a fixed intensity below the damage threshold, then accurate evaluation would be represented by a close correlation between intensities of mechanical energy and pain. *Sprain* is drawn from common actual cases so it is clear that in the presence of tissue damage at least evaluations are mostly inaccurate. The variability of the relationship between the intensity of energy and pain is explained by the difficulty of evaluating whether energy is noxious.

When tissues are undamaged subjects are usually fairly consistent in the mechanical demands they impose on the tissues. Even if someone has a sedentary lifestyle barring a hard game of squash on Friday evenings there is a consistency to their demands. It can be said that the subject is used to the things she does. By this I do not mean that she is psychologically accustomed to her physical life, I mean that her sub-personal mechanisms are adapted to this routine. *FS* is part of this adaptation. In particular *CI*, which constantly monitors sensory input, does not evaluate many of the patterns of this routine as being worthy of response. Tissue damage dramatically changes this picture.

Suddenly the damage threshold changes, if there were no change to the evaluation of energy by *CI*, then further damage would be a certainty. Clearly, *CI* does evaluate energy differently because we experience pain doing things that would have been pain free prior to injury (*Sprain* is an example of this). Now it is easy to assume that in

such circumstances *CI* is accurately evaluating the threat; that pain is correlated with noxious intensities of energy. It is certainly normal to experience quite severe pain after twisting an ankle (spraining a ligament) so that weight-bearing is extremely painful. But there is little reason to think that *CI* is making *accurate* evaluations in such circumstances unless it can accurately evaluate the effect that tissue damage has on a tissue's ability to cope. This would require *CI* having accurately evaluated the pre-morbid damage threshold, accurately evaluating the impact of tissue damage on that pre-morbid threshold (i.e. the accurate evaluation of the current damage threshold) and accurately evaluating the current demand imposed by energy. And all of this would have to be done in a short space of time. There is no evidence that any such accurate evaluative mechanisms exist. Instead, post-injury pain is the product of a crude mechanism by which the stimulus thresholds of sensory neurons are dramatically lowered so that *CI* tends to over-protect. Subsequently, *CI* has to learn what can and cannot be done. It does this through *comparison*. *Sprain* exemplifies this comparative process.

When the subject has been sitting for a while, the mechanical energy being absorbed by the tissues is slight. When she puts her foot to the floor and rises there is a dramatic increase in the force being imposed on the relevant tissues and *CI* evaluates that this energy is worthy of a considerable response. The reason it makes this evaluation is not because a particular intensity threshold has been reached, it is because of the relative difference in the input associated with the resting and standing demands; i.e. *CI* makes a comparison. One of the crucial factors in this comparison is the length of time the subject had been inactive. Limping around eases her pain because there are no

dramatic increases in input. If she suddenly tried to run or jump or the demands of walking were so great that the damaged tissue released algogenic substances the afferent barrage would suddenly (in the former case) or rapidly (in the latter case) increase prompting a response from *CI*. When the subject sits for a few moments before rising again, the reason that she feels little pain is obviously not because of the difference between the inputs associated with inactivity and standing because this difference is the same as the first time she rose from the chair. The difference is explained by the length of time she was sitting prior to standing in each case. The first time the comparison was with a long period of sitting; *CI* has little to go on so it tends to over-protect. The second time the comparison is with (successful) activity about, and does not over-protect to the same extent.[32]

It is important to note that this account of *Sprain* does not explicitly refer to the accuracy of the evaluations carried out by *CI*. While the evaluations carried out by *CI* with respect to chronic low back and neck pain are assumed to be inaccurate, those associated with acute injuries like sprains are assumed to be accurate. My view is that the latter are rarely accurate if accuracy is taken to refer to some objective notion of threat. The concept of accuracy needs refining in this context. The issue is whether *CI* and *CO* function adequately as constituents of the overall nocifensive function of *FS*. If they do then they are accurate enough for purpose. Given the nature of the function of *FS* it is to be expected that *CI* tends to err on the side of caution. Accuracy would be ideal, but it is not an absolute standard by which the occurrence of pain should be

[32] To say that *CI* is *accurately* evaluating threat the second time she stands would be nothing more than an assumption.

judged. In short, given the biological resources a weak correlation between pain and noxious intensities of energy is the norm it is not an anomaly.

Inaccuracy to the extent that the subject is disadvantaged is by contrast a very real problem. Some cases of chronic pain are examples of gross inaccuracy. In these cases pain persists over time because of maladaptive plasticity. While the comparisons that explain *Sprain* are explanations of short term fluctuations of the relationship between intensities of energy and pain, the problem for those with chronic problems is that long-term memory is affecting the evaluations carried out by either or more likely both *CI* and *CO*. In effect, the chronic pain state represents the *status quo* because of changes to synaptic architecture. So the relationship between intensities of energy and pain cannot change without changes to this architecture.[33] This is why some chronic cases are such difficult medical problems.

The purpose of this chapter has been to tie up some loose ends derived from the constitutional problems posed in P2 and to explain P4-P6. With respect to the former I have defined pain for the purposes of the remains of my thesis, but ultimately left the question of the scope of 'pain' open because it has no important bearing on my thesis. I have also addressed the issue of recognition. Although the explanation of some cases of noxious intensities of energy in the absence of pain are relatively easily explained in terms of an evaluation (by *CO*) of whether, what and how much a nocifensive response is appropriate given the context, other problems are more taxing. The insight provided by the realisation that the discrimination of noxious intensities of energy is

[33] See Sandkuhler (2000), for more on the influences of learning, memory and plasticity on pain.

not a simple peripheral affair has been invaluable. The explanation of these other problems is bound up with complexities of evaluating (by *CI*) which stimuli represent a threat. Chronic pains are explained by the adverse effect that neurological plasticity can have on *CI*, and everyday occurrences of injury in the absence of pain are explained by patterns of action potentials that *CI* does not recognise as worthy of response. Although some of the detail of my account of *CI* and *CO* has been speculative, I am not aware of any plausible alternatives to my account of *Sprain*. This highlights the trial and error basis of the evaluations carried out by *CI* in the presence of tissue damage.

6.4 CONCLUSION

The challenge I posed at the beginning of this thesis was to explain several constitutional (P1 and P2), functional (P3) and empirical problems (P4-P6) while remaining consistent with science. These problems are intimately connected. Constitutional nature and function must be consistent. Errors here are bound to create explanatory problems elsewhere; with science or the sort of empirical problems I have set out under P4-P6, for example. The composite structure of pain is problematic precisely because it has three aspects: its two constituents considered independently of one another and the composite itself, pain.

Considered separately, there is very good reason to believe that the constituents of a pain, a token affectively neutral phenomenal (a token of *Q*) quality and negative affective tone (*U*) *are* perceptual and motivational because the former has perceptual

features and the latter has motivational features. So superficial consideration of *Q* and *U* favours mixed perceptualism/motivationalism because a perceptual account of *U* and a motivational account of *Q* are on the face of it implausible. Implausible that is until it is realised that the explanatory task is not one of explaining the features of *Q* or *U* in contrary functional terms. The task concerns *Q* and *U* as functional constituents *of pain*.

Pain undoubtedly has a motivational function. Those unable to experience pain do not behave in ways that are sufficiently effective to prevent serious injury. But this "undoubted" function does not appear to be entirely consistent with its nature. Pain has perceptual features derived from *Q*. Mixed theorists have a ready explanation for this; the content of *Q* fulfils a perceptual function in pain. Specifically, a pain is constituted by a token of *Q* that represents a noxious intensity of energy and negative affective tone (*U*) so subjects are motivated by the noxious energy represented by a token of *Q*. This account relies upon the assumption that *Q* is constituted by phenomenology that is unique to pain. A denial of this assumption is the key argument in my thesis.

In terms of neurology a stimulus is something that can be detected by a sensory receptor. A noxious stimulus cannot be detected by any sensory receptor so it is not a genuine stimulus and pain is not subserved by a specific sensory system. This conclusion has two important consequences: first, pain is partly constituted by the same qualities as innocuous thermal and mechanical sensations; and second, the discrimination of noxious intensities of energy is a complex process involving the

central nervous system. The former has significant implications for the understanding of the constitution and function of pain. The latter provides a means of explaining the most difficult empirical problems.

Suddenly the mixed theory is much less appealing. If *Q* represents energy, then what represents *noxious* energy? If *U* is taken to represent the noxious nature of energy the mixed theory slides into perceptualism and it is vulnerable as a consequence. If the position is that we are motivated by energy, why is it that we are being motivated by some token experiences that represent energy and not others? The reason is not given by the content of the experience. Now the problems posed by P4-P6 and the intuition that we are motivated by pain and not the content of pain seem more significant. What is more, one of the obstacles to motivationalism, the perceptual features of pain, can be convincingly explained – pain has perceptual features because the thermal and mechanical qualities that constitute *Q* have evolved to fulfil a function as thermal and mechanical sensations.

My version of motivationalism, near-motivationalism is derived from these aspects of pain. On my account, pain is a product of a system (*FS*) that has a broad nocifensive function. *FS* is partly constituted by mechanisms that function to discriminate noxious energy (*CI*) and evaluate responses (*CO*). When *CI* evaluates that energy is worthy of a response, and *CO* evaluates that pain is an appropriate response given the context, *FS* attaches negative affective tone (*U*) to a token thermal or mechanical phenomenal quality (a token of *Q*) that is causally related to the energy that *CI* is evaluating as worthy of response. This has the effect of making an affectively neutral phenomenal

quality feel as if it is intrinsically unpleasant and the subject experiences pain. In most circumstances this attracts an aversion which motivates behaviour aimed at ridding the subject of the pain. This prevents injury because the token of *Q* is causally linked to the energy that *CI* has evaluated to be worthy of a response.

Problems P4-P6 are explained by the evaluations either carried out by *CI* or *CO*. Hitherto, the only available explanation for problem cases has been *CO* and maladaptive plasticity. The difficulty of discriminating threat has not been a feature of any philosophical or scientific accounts because the (scientific) concept that noxious energy is discriminated peripherally has been unquestioned. It is partly for the reason that no specific research has been carried out that my account of *CI* and *CO* is undeveloped and to a certain extent speculative. The other reason is that the development these evaluative components of nocifense represent a major undertaking, which is probably in excess of the work I have undertaken for this thesis. This is an area that merits future research.

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